MEDICAL PROCEEDINGS

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Medical Proceedings · Mediese Bydraes

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Vol. III, No. 1, 1956

CONFERENCE ON NUTRITION IN INFECTIONS

Under the auspices of THE NEW YORK ACADEMY OF SCIENCES (Section of Biology)
2 East Sixty-Third Street, New York 21, New York

CLINICAL REPORT

NEW YORK MEDICAL COLLEGE, NEW YORK CITY: RECOVERY FROM ILLNESS, INJURY, SURGERY RELATED TO NUTRITION - Statistically significant studies indicate that "good nutrition is important for optimal resistance to infection . . . maximum antibody production . . . rapid wound healing . . . tissue synthetic processes . . . correct hormone and enzyme activity . . . " normal blood protein levels, osmotic pressure and hematopoiesis. Infection raises requirements "for most nutrients because of the increased metabolic rate secondary to fever and toxicity and the physiologic changes produced in the tissues by the stress of the disease." All patients with infection require nutritional fortification. The "combination of an antibiotic with vitamins in a single capsule" assures "adequate intake of those vitamins which are especially needed in stress situations under treatment with antibiotics . . . " may speed convalescence and reduce incidence of complications. "In some instances of acute illnesses, injury or surgery, intensive nutritional therapy may be the deciding factor in the outcome. The course of chronic disease as well as the duration of convalescence from acute pathology may be accelerated or retarded as a result of the diet pattern. Nutritional therapy . . . must be correlated with other . . . therapy of the illness."

Halpern, S. L.: Critical Evaluation of the Role of Nutrition in the Prophylaxis and Treatment of Disease.

UNIVERSITY OF MINNESOTA, MINNEAPOLIS, MINN.: ALL METABOLIC PROCESSES INFLUENCED BY NUTRITION - Since nutrition influences all metabolic processes, it is clear that the nutritional status of the individual is a determining factor in the development and character of disease, regardless of etiology. Antibiotics and chemotherapy have "produced great victories in the fight against infectious diseases, but there is still a 'cold war' against the microbial world, and in this 'cold war' . . . nutritional knowledge has an important role to play."

Grande, F.: Discussion of Dr. Halpern's paper, "Critical Evaluation of the Role of Nutrition in the Prophylaxis and Treatment of Disease."

HARLEM HOSPITAL, NEW YORK CITY: NUTRITIONALLY FORTIFIED BROAD SPECTRUMS "MOST EXCELLENT" - The combination of broad-spectrum antibiotics (Terramycin* [brand of oxytetracycline] and Tetracyn* [brand of tetracycline]) with vitamins is a decided advance "in the total care of the young, the aged and the debilitated . . . " Infections disrupt nutritional equilibrium by reducing available nutrients "through decreased absorption, decreased utilization, increased demand, and increased loss of nutritional elements." Nutritionally fortified antibiotics supply the patient with the nutrients to combat the stress† situation while fighting the pathogenic invaders. In studies conducted at Harlem Hospital, the administration of Terramycine* SF* (brand of oxytetracycline with vitamins) or Tetracyn* SF* (brand of tetracycline with vitamins) to surgical patients aged 7 months to 81 years proved "eminently satisfactory, with many recalcitrant infections being brought promptly under control." Wound healing and convalescence proceeded without complications. "No allergic or side reactions were observed . . . Despite malaise, pain, and other manifestations of severe illness, the patients' appetite and general condition were satisfactory." Dosage: 250 mg. antibiotic-vitamin combination four times daily for adults, proportionately less for children.

Prigot, A.: Fortified Broad-Spectrum Antibiotics as an Adjunct in Surgical Treatment of the Young, and the Aged and Debilitated.

UNIVERSITY OF PITTSBURGH, PITTSBURGH, PA. TERRAMYCIN SF AND TETRACYN SF EFFECTIVE, EVEN BETTER TOLERATED - Terramycin SF and Tetracyn SF have given clinical results comparable to those achieved with the antibiotics alone and possibly with a lower incidence of side effects. Among 53 patients with various infections treated with Tetracyn SF, those with acute clinical illness resulting from either indefinable or sensitive pathogens "responded satisfactorily within twenty-four to forty-eight hours." A patient with refractory Proteus urinary tract infection became asymptomatic, "afebrile and with a diminishing pyuria in spite of persistent positive urine cultures . . ." To evaluate drug tolerance, Tetracyn SF was given in doses of 1.5 to 2.0 Gm. daily for 5 to 14 days. " . . . we dealt with a paucity of complaints in view of dosage and length of time of drug administration." Clinical and experimental data support "a belief in the value of a high protein intake in conditions of stress. As a corollary, it may be stated that those vitamins known to be of importance as units in enzymes concerned with protein metabolism must also be provided in increased amounts because of their greater utilization in this function." In turn the complex interrelationships of the various essential vitamins involved in metabolic processes suggest "that the optimal effect of a single vitamin given in large doses cannot be exerted unless the supply of all others is proportionally increased."

Milberg, M. D., and Michael, M., Jr.: Antibiotics and Nutrition in Infections.

INSTITUTE FOR METABOLIC RESEARCH, HIGHLAND ALAMEDA COUNTY HOSPITAL, OAKLAND, CALIF.: PITUITARY-ADRENAL FUNCTION "MAJOR" FACTOR IN RESPONSE TO INFECTION - Diet "high in protein, adequate in calories, moderate in carbohydrates; and very adequate in ascorbic acid and members of the B-complex, with particular reference to pantothenic acid, is compatible with optimal ACTH-adrenal steroid production . . . which constitutes part of the major defense machinery of the body . . . A voluminous literature attests to the importance of the pituitary-adrenal axis in response to stress, whether the stress be in the form of infection, operative trauma, thermal damage, etc." In the patient with increased adrenal cortical activity as a result of infection "and even more in the individual with infection who requires supplementary corticoid therapy . . . " it is advisable to prescribe a diet "high in protein, relatively high in calcium and phosphorus, low in carbohydrate, adequate

*Trademark of Chas. Pfizer & Co., Inc.
†Stress, as referred to herein, is a state in which the metabolic demands of the body are increased as a result of infection.

in calories, low in sodium, and very high in potassium" plus four to five times the normal requirement of essential vitamins and unsaturated fatty acids. When corticoid therapy is mandatory in patients with initially poor nutritional status, " 'prophylactic' broad-spectrum antibiotic therapy should always be co-administered" in addition to the nutritional program outlined. "The increased hypersusceptibility to infection which characterizes malnourished patients who are receiving corticoids is probably attributable to a variety of factors, which include prominently the inhibition of the inflammatory reaction; diminished production of antibodies, partly as the result of the increased protein catabolism: (this statement applies only to malnourished patients); increased tissue edema, with consequent tissue anoxia, and diminished cellular resistance."

Kinsel, L. W.: Nutritional and Metabolic Aspects of Infection.

LOUISIANA STATE UNIVERSITY, NEW ORLEANS, LA.: DIET PRIMARY FACTORS IN HOST RESISTANCE TO INTESTINAL PARASITIC INFECTION - While intestinal parasites may reside harmlessly in the gastrointestinal tract, alteration in diet may be the trigger mechanism in the lowering of host resistance and the production of gross pathology by the parasites. "The adjustments between parasite and host are often extremely delicate and only slight changes may mean the difference between damage and no damage to the host tissue . . ." Vitamin deficiency can act in a twofold manner that is detrimental to the host (1) by favoring the parasite and (2) by interfering with host resistance. The administration of nutritionally fortified antibiotics to patients with amebiasis or helminthiasis corrects dietary imbalance while eradicating the parasitic infection. In studies now in progress in a pediatric institution Terramycin* SF* (brand of oxytetracycline with vitamins) and Tetracyn* SF* (brand of tetracycline with vitamins) are being used with excellent results and outstanding tolerability. Some patients have noted increased appetite and weight gain as well.

Frye, W. W.: Nutrition and Intestinal Parasitism.

MICHAEL REESE HOSPITAL, CHICAGO, ILL.: VITAMIN A STORES LARGE, YET BLOOD AND LIVER LEVELS LOW IN INFECTION - In experimental infection, as in human infection, blood and liver vitamin A are decreased, possibly because of impaired conversion of B carotene to vitamin A. Relatively long period of deprivation (three months) is necessary before influence of vitamin A deficiency on susceptibility to infection becomes significant. "This is most likely due to the fact that large amounts of vitamin A are stored in the liver" in the human (with exception of infant). Despite this vitamin A reserve, there is some clinical evidence that supplementary administration of the vitamin may have a beneficial effect on the course of infective processes.

Kagan, B. M.: Observations on Infection and Vitamin A.

UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA, PA.: "OPERATION TENDS TO ACCENTUATE PROTEIN DEFICIENCY" - and the " . . . effect of hypoproteinemia is to increase the chance for a variety of operative and post-operative difficulties." Restoration of nutritional deficits in surgical patients is "of paramount importance." In a study of 102 surgical cases, the incidence of infection was found to be higher in the hypoproteinemic group of patients than in the control group. "It is thus of tremendous importance to find ways of giving surgical patients sufficient foodstuffs by the simplest routes available, to supply their unusual needs."

Rhoads, J. E.: Nutritional Problems of Surgical Patients.

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JOHANNESBURG GENERAL HOSPITAL, UNION OF SOUTH AFRICA: INFECTION "INTENSIFIES POOR NUTRITION" - In the presence of "borderline nutrition, the onset of acute infection . . . may predispose to nutritive failure, which . . . may be responsible for affecting adversely the patient's well being and may endanger his life. Early recognition of his acute dietary deficiency state and replacement therapy are called for . . . The mechanism for the development of nutritional failure in the presence of infection can be explained possibly on the basis that the invading organism depletes the host of its low stores of available nutrients; and also perhaps because the patient's level of metabolism has been raised by the infection, thereby increasing the requirements of essential nutrients beyond the body's available supply." In fulminating infections, the administration of adrenal hormones may prove lifesaving. In patients exhibiting exaggerated inflammatory reactions and in cases of "acute overwhelming" infection featured by "impending shock and circulatory collapse with threatened renal shutdown, with disturbed electrolyte and water balance . . . the inflammatory process generally is not that benign, protective, laudable phenomenon it has hitherto been thought to be, and when needs be, it must be curbed. This is achieved best in our present state of knowledge by appropriate antibiotic therapy supported by the judicious use of adrenocortical hormones and the maintenance of good nutrition."

Suzman, M. M.: Discussion of Dr. Kinsell's paper, "Nutritional and Metabolic Aspects of Infection."

NEW YORK MEDICAL COLLEGE AND FLOWER AND FIFTH AVENUE HOSPITALS, NEW YORK, NEW YORK: VITAMIN FORTIFIED BROAD SPECTRUMS "SINE QUA NON" IN TROPICAL MEDICINE - "Malnutrition and infections in the tropics are inseparably linked . . . " Malnutrition predisposes to infection and conversely infection is a "conditioning factor which aggravates the nutritional deficiency." The combination of infection and deficiency results in a stress situation that "can cause adrenal cortical changes, and produce changes in those organs with high metabolic activity, especially the small intestine and liver." Terramycin* SF* and Tetracyn* SF* are therefore especially valuable in in tropical countries where malnutrition is a common problem.

Tropical ulcer and ulcerous lesions of late yaws and intestinal amebiasis "heal more rapidly" when treated with Terramycin SF and Tetracyn SF. These vitamin fortified broad-spectrum antibiotics "provide a safe balanced combination of these essential substances - a fact that may be critically important in the treatment of infections when they are associated with kwashiorkor . . ." The vitamins "exhibit no patent inhibitory effects on the antiparasitic activity of either antibiotic . . . the absence of notable toxic reactions in 134 cases of bacterial, protozoan and metazoan infections treated with Terramycin SF and Tetracyn SF in New York City as well as in Haiti is noteworthy . . . "

Loughlin, E. H., and Mullin, W. G.: Certain Aspects of Deficiency Diseases of the Tropics and Treatment of Some Related Infections.

CAIRO UNIVERSITY, CAIRO, EGYPT: DIET MAY AFFECT AMEBIC INVASION - In certain areas of the world where the diet consists mainly of carbohydrate food (maize), the incidence of intestinal and hepatic amebiasis seems to be no greater in malnourished than in well nourished individuals. This has been attributed to the fact that encystment of amebae does not readily occur when the stool is acid.

Mousa, A. H.: Discussion of Dr. Frye's paper, "Nutrition and Intestinal Parasitism."

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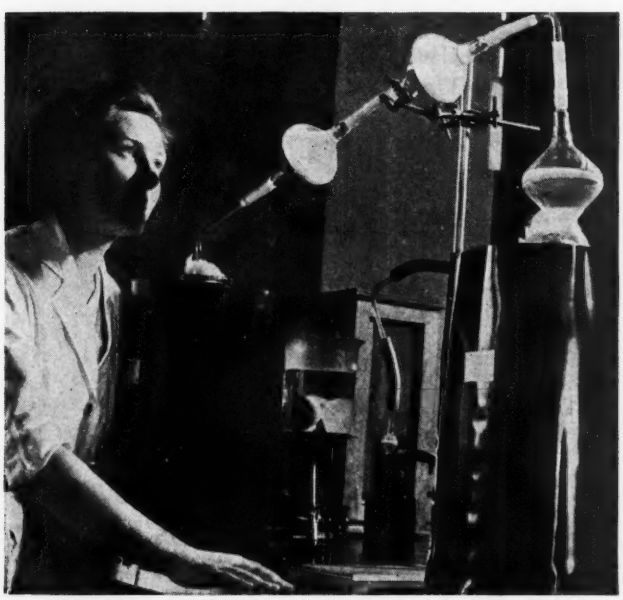

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REDAKSIONEEL · EDITORIAL

EX AFRICA SEMPER ALIQUID HORRENDI

VASSTELLING VAN EN BEHEER OOR PRIVATE DOKTERSREKENINGS

Deur die oplegging op private mediese praktisyns van 'n stelsel ten gevolge waarvan die gelde wat verskillende soorte praktisyns vir hul dienste mag vra, voorgeskryf en beperk, en hul verdienste gevolglik aan bande gelê word, beklee Suid-Afrika oënskynlik 'n unieke posisie onder die beskaafde volke van die wêreld. Voor 1954 is pogings aangewend om geskille oor private doktersgelde te besleg deur middel van artikel 80 van die Wet op Geneeshere, Tandartse en Aptekers, maar die resultate van hierdie pogings kan slegs as ontstellend bestempel word. 'n Wysiging van die Wet in 1954 het egter 'n splinternuwe bevoegdheid aan die Mediese Raad verleen vir soverre dit die ondersoek van klagtes oor sodanige private doktersrekenings betref. Die lompe en tydrowende masjinerie wat nou in die lewe geroep is en elders in hierdie uitgawe verduidelik word, bied die ligsinnige pasiënt 'n geleentheid om die betaling van sy rekening 'n hele ruk lank uit te stel deur bloot 5% van die bedrag wat in dispuut is, te deponeer. Daar word van hom verlang om sy dokter vooraf te nader, maar dit weer nie noodwendig die volgorde van gebeurtenisse af wat reeds aan die gang gesit is nie.

Hierdie gebeurtenisse word, weliswaar, nie meer outomaties in die openbaar strafregtelik ondersoek nie—'n stelsel wat onregverdiglik op praktisyns kon ingewerk het, en dit ook ge-

ASSESSMENT AND CONTROL OF PRIVATE MEDICAL FEES

South Africa appears to be unique amongst the civilized nations of the world in having imposed on private medical practitioners a system which seeks to prescribe and limit what different categories of practitioners may charge for their services, and consequently what they may earn. Before 1954 attempts were made to control disputes about private fees by section 80 of the Medical, Dental and Pharmacy Act, with what can only be described as appalling consequences.

But an amendment to the Act in 1954 gives the Medical Council a new rôle in the investigation of complaints about such private fees. The ponderous and time-consuming machinery now set up (and published elsewhere in this issue) allows a frivolous patient to hold up payment of an account for a prolonged period merely by depositing 5% of the amount in dispute. He is required to make a prior approach to the doctor, but this does not necessarily stave off the sequence of events set in train.

True, this sequence is no longer automatically the subject of a public penal inquiry, a system that could and did operate with such injustice to practitioners before section 80 was amended. Hence this is a good feature of the amendment, long overdue.

But now the assessing body (whose members may or may not have had adequate, or

doen het, voordat artikel 80 gewysig is. Dit is gevolglik 'n goeie kenmerk van die amendement, en moes lankal reeds ingevoeg gewees het.

Maar nou word die vasstellingsliggaam (wie se lede miskien voldoende, onvoldoende, of glad geen kennis van 'n private mediese praktyk het nie) kragtens wet gemagtig om die maksimum-bedrag te bepaal wat 'n geneesheer, *volgens die mening van die vasstellingsliggaam,* moes gevra het. Die pasiënt sowel as die mediese praktisyn kan vertoë (maar alleen skriftelik) oor die onredelikheid of andersins van die rekening voorlê. Maar sodanige vertoë kan egter nie op enige wyse aan kruisverhoor deur enigeen van die partye onderwerp word nie.

Die besluit van die vasstellingsliggaam is finaal en bindend, hoe arbitrêr dit ook al mag wees, en die mediese praktisyn sal feitlik 'n soort boete moet betaal as daar 'n verskil van meer as 10% tussen sy en die Raad se skatting is. Gesonde verstand kom in opstand teen die idee dat 'n rekening buitensporig is bloot omdat dit, numeriek-gesproke, 'n tarief oorskry (met hoe min ook al) wat die Raad bereid is om as aanneemlik te beskou.

Nadat die bedrag van die rekening bepaal is, kan die Raad ondersoek instel na die gedrag van die mediese praktisyn (artikel 80 *bis* (18)). Daar heers duisterheid oor hoe dit presies gedoen sal word. Indien die Raad sou kan voortgaan, kan hy (uit hoofde van die dissiplinêre magte wat kragtens Hoofstuk IV van die Wet aan hom toevertrou is) die betrokke geneesheer skuldig bevind aan onbetaamlike of skandelike gedrag. So 'n bevinding stel die geneesheer dan bloot aan die baie swaar strawwe wat die Raad verplig sal wees om op te lê.

In hierdie sake kan geen appèl teen die feitebevindinge van die Mediese Raad by die howe aangeteken word nie. Dis derhalwe moontlik dat die beperkte en onbevredigende hersieningsregte wat tans tot beskikking van die mediese beroep is, nog verder versnipper en selfs heeltemal waardeloos gemaak kan word deur die wysigings wat in 1954 in die Wet aangebring is.

Die huidige wetgewing kan dus meebring dat 'n gewettigde meningsverskil oor 'n rekening in bepaalde omstandighede as onetiese gedrag vertolk word. Maar dit sal wangedrag wees wat kunsmatig geskep is—m.a.w. geen morele beginsels sal op die spel wees nie. Die mediese professie sal moeilik oortuig word dat nie-etiese geskille in sekere omstandighede any, experience of private medical practice) is empowered by law to determine the maximum amount which, *in its opinion,* the practitioner should have charged. Both the patient and the practitioner may make submissions (in writing only) about the unreasonableness or otherwise of the fee; but these submissions cannot be tested by cross-examination in any way by any of the parties concerned.

The decision of the assessing body is final and absolute, even if it is arbitrary, and the practitioner may have to pay what is virtually a fine if he and the Council differ in their estimate by more than 10%. Common sense revolts against the concept that a fee is excessive merely because it exceeds numerically, by no matter how modest a margin, a tariff which the Council may be prepared to find acceptable.

After determining the fee the Council can hold an inquiry into the conduct of the practitioner (section 80 *bis* (18)). There is a certain obscurity about how this will now be done. If the Council can proceed, it may (in terms of the disciplinary powers vested in it by Chapter IV of the Act) find him guilty of improper or disgraceful conduct. Such a finding renders him liable to the very severe penalties which the Council may then be obliged to impose.

In these matters there is no right of appeal to the Courts from findings of fact by the Medical Council. The 1954 amendment may well have pared down even more severely the profession's limited and unsatisfactory rights of review, if not to have rendered them nugatory.

The present legislation may thus ensure that a legitimate difference of view about a fee in a particular set of circumstances may be construed as an unethical act. But this is an artificially created act of misconduct, not rooted in any moral principle. The profession will not readily be convinced that non-ethical disputes should be regarded as unethical acts, especially when both aggrieved parties have all the potent remedies provided by common law for the settlement of such issues.

The 1954 amendment is presumably intended to facilitate the adequacy of inquiries into disputed fees. Whether it is a just mechanism for settling such disputes, remains to be seen. What has been created is power for the Council to practise a form of price control of fees completely foreign to its proper concern with ethical standards of professional conduct.

as onetiese dade bestempel kan word, veral wanneer albei partye tot die geskil al die kragtige hulpmiddele van die gemeenreg vir die beslegting van sodanige dispute tot hul beskikking het.

Die 1954-amendement is vermoedelik bedoel om die ondersoek van 'n rekening waaroor daar geredetwis word, doelmatiger te maak. Of dit 'n regverdige middel vir die beslegting van sodanige geskille skep, sal nog besien moet word. Wat wel gedoen is, is om bevoegdheid aan die Raad te verleen om 'n soort prysbeheer oor doktersrekenings uit te oefen—iets wat glad nie tuishoort by die Raad se heeltemal behoorlike belangstelling in die etiese maatstawwe van professionele gedrag nie.

Die ekonomiese sy van 'n mediese praktyk is nie iets wat na regte by die Mediese Raad tuishoort nie, en die swaar las wat die beroep reeds moet tors vir sover dit die finansiering van die Raad se werk betref, word nou onnodig swaarder gemaak.

KONTRAKTARIEWE IN HUL JUISTE PERSPEKTIEF

Omdat die Mediese Vereniging uiteindelik toegegee het (heeltemal verkeerdelik, meen ons) aan die herhaaldelike versoeke van die Raad om 'n tarief van doktersgelde, kan daar aangeneem word dat die vasstellingsliggaam mag besluit om hierdie tarief (wat hom in elk geval nie bind nie) te raadpleeg en dit te gebruik as 'n leidraad tot private doktersgelde.

Die aard van die tarief verdien derhalwe sorgvuldige ondersoek, want dit kan die statutêre liggaam beïnvloed in sy beslissing oor die lot van 'n mediese praktisyn.

In soverre 'n tarief van die hierbo beskrewe tipe as maatstaf gebruik kan word, moet daar in gedagte gehou word dat dit gebaseer is op 'n skaal wat spesiaal ontwerp is vir diegene wat nie ryklik met wêreldsgoed bedeel is nie. Die mediese beroep het nog altyd hulp verleen aan diegene wat hulp nodig het, al kon die pasiënt ook nie die volle private gelde bekostig nie. Dus, toe gesonde burgers hulle saamgeskaar en voorsorgsmaatreëls begin tref het vir die dag wanneer hulle deur siekte oorval word, het ons kollegas (besiel deur hul geliefde Hippokratiese tradisie) hulle sonder die minste aarseling bereid verklaar om die verenigings wat mediese hulp vir hul lede wou hê, op dieselfde wyse te help as wat hulle nog altyd individuele pasiënte wat miskien in finansiële moeilikheid verkeer, gehelp het. Die doktersgelde, neergelê in die tariewe wat op Mediese Hulpverenigings van toepassing is, het der-

The economics of medical practice is not the proper study of the Medical Council and the heavy burden which the profession already shoulders in financing the work of the Council is being increased unduly.

CONTRACT PRACTICE TARIFFS IN PROPER PERSPECTIVE

Because the Medical Association has ultimately acceded (mistakenly, we believe) to the repeated requests of the Council for tariffs of fees, it can be presumed that the assessing body may elect to consult these tariffs, which (in any event) are not binding on it, as a guide to private fees.

The nature of the tariffs therefore deserves careful scrutiny, because they may influence the statutory body in determining the fate of a practitioner.

In so far as a tariff of the type described may be used as a guide or a yardstick, it must be remembered that it is based on a scale of fees evolved for those not blessed with an abundance of worldly things. The profession has always helped those who needed their care, even though these patients could not afford full private fees. So, when citizens banded together when they were well, to insure against risks when they were ill, our colleagues (inspired by their devoted Hippocratic tradition) unhesitatingly did for the societies seeking medical aid for their members what they had long done for individual patients when these were in financial difficulties. The fees enshrined in the tariffs applicable to Medical Aid Societies therefore grew up as a special contractual arrangement with groups who felt they were in need.

The argument that the fee was guaranteed by the Society and that there would therefore be no bad debts could only have warranted a reduction by about 5–10% and not 33⅓% (as at present) on the assumption of the fiction that the tariff is related to private fees by a simple arithmetical formula.

The assurance that fees would be paid promptly has not, in fact, prevented delays in the settlement of accounts.

The argument that patients (who might otherwise have gone to public hospitals) would be diverted to private practitioners neglects the fact that these patients no longer waste hours or even days in queues at Out Patient Departments, with consequent loss of earnings for the patient, inevitable and undesirable delay in diagnosis and treatment, as well as wasteful loss of man-power in industry and

halwe ontstaan as 'n spesiale kontraktuele reëling met groepe wat gemeen het dat hulle behoefte aan sodanige hulp het.

Die argument dat betaling van die rekening deur die onderstandsvereniging gewaarborg word, en dat daar derhalwe geen slegte skulde kan wees nie, kan as regverdiging dien vir 'n vermindering van tussen 5 en 10 persent, en nie 33⅓ persent soos op die oomblik nie, en alleen as ons bereid is om te glo aan die verdigsel dat daar 'n eenvoudige rekenkundige formule bestaan wat die verband aandui tussen hierdie tariewe en private doktersgelde.

Die versekering dat rekenings stiptelik betaal sal word, het in die praktyk nie vertragings met die vereffening van sodanige rekenings uitgeskakel nie.

Daar word ook geredeneer dat pasiënte, wat andersins miskien na openbare hospitale sou gegaan het, nou na private mediese praktisyns kom. Maar hierdie argument hou geen rekening met 'n belangrike feit nie, nl. dat hierdie pasiënte nie langer ure en selfs dae hoef te verkwis deur tou te staan by die Buitepasiëntafdelings van ons hospitale nie, met die daaruit voortvloeiende verlies van verdienste vir die pasiënt, die onvermydelike en onwenslike vertraging met diagnose en behandeling, asook die verkwisterige verlies van arbeidskrag wat dit vir ons handel en nywerhede meebring. Nog minder hou dit rekening met die feit dat pasiënte wat aan 'n mediese onderstandsvereniging behoort, presies dieselfde aandag en belangstelling as 'n private pasiënt ontvang, en dat hulle die dokter dag en nag kan hiet en gebied.

Bloot op ekonomiese gronde is daar derhalwe geen geldige rede waarom 'n lid van 'n Mediese Hulpvereniging nie die volle private doktersgelde, waarteen hul lidmaatskapbydraes hulle behoort te verseker, moet betaal nie. Burgers wat hulle teen die verskillende risiko's van die lewe wil vrywaar, gaan allerhande soorte versekerings aan. En tog is dit net ten opsigte van siekte dat daar van die professie wat die nodige diens verskaf, verwag word om daardie diens te subsidieer. Net so min as wat kan verwag word dat dokters die lewensversekeringspremies van die lede van Hulpverenigings moet subsidieer kan verwag word dat hulle sulke lede se bydraes tot Hulpverenigings moet subsidieer.

Selfs die mening dat doktersrekenings verminder moet word omdat armer lede minder, en ryker lede meer betaal, is nie gegrond nie, want een van die grootste verenigings in die land (die voormalige Nasionale Mediese Hulp-

commerce; and that Medical Aid Society patients receive the care and concern that all private patients receive, the doctor being at their beck and call night and day.

Merely on the economic merits there is therefore no valid reason why members of a Medical Aid Society should not pay full private fees, for which their membership contributions could insure them. Citizens seeking protection against various risks in life take out all kinds of insurance. Yet it is only in respect of illness that the profession providing the service is expected to subsidize it. There is no more reason for doctors to subsidize Medical Aid Society contributions than there is for them to subsidize the life insurance premiums of the members.

Even the view that medical fees should be reduced because poorer members pay less and richer members pay more is without substance because one of the biggest societies in the country (the former National Medical Aid Society) had, for practical purposes, no sliding scale of contributions.

The enrolment of Associate Members also brings into a society's fold high-income groups which do not otherwise qualify for admission. The extraordinary position can in fact arise in which practitioners treat at reduced fees patients whose income tax assessments may exceed the actual income of the practitioners concerned.

Despite all the sound arguments against a reduced tariff, the medical profession has never hesitated to assist those who needed their help. This is the real and moral justification for the reduced tariff for groups such as the Medical Aid Societies. Practitioners will not readily deprive themselves of their right to an unfettered discretion in deciding to help those who need help, even though the assistance they give operates to their own economic disadvantage. *But this charity must not be abused to impose a ceiling on private fees.* It is therefore important to remember that the special tariffs were never computed actuarially. They take no account of the rapid and considerable increases in the cost of living against which a great multitude of citizens is cushioned and protected by law in the public service, in industry and commerce.

The price that the medical profession pays in cash for maintaining the welfare of a section of the community is well brought out by the enormous surplus accumulated by the Workmen's Compensation Commissioner. Despite rebates to industry running into hundreds of thousands of pounds, the reserve

vereniging) het in die praktyk feitlik nie so iets soos 'n wisselende skaal van bydraes.

Daarbenewens het die toelating van geassosieerde lede sekere hoë-inkomste-groepe, wat andersins nie die nodige kwalifikasies vir lidmaatskap sou besit het nie, binne die bestek van verenigings gebring. Die buitengewone toestand kan dus ontstaan dat 'n dokter 'n pasiënt wie se belastingsaanslag die totale jaarlikse inkomste van die betrokke dokter oorskry, teen 'n verminderde tarief moet behandel.

Ten spyte van al die gegronde besware wat teen 'n verminderde tarief ingebring kan word, het die mediese beroep nog nooit geaarsel om hulp te verleen aan diegene wat hulp nodig het nie. Dis die eintlike en morele beweegrede vir die vermindering van tariewe vir groepe soos Mediese Hulpverenigings. Mediese praktisyns sal nie maklik afstand doen van hul reg om na goeddunke hulp te verleen aan hulpbehoewendes nie—al strek sodanige hulpverlening ook tot ekonomiese nadeel van die betrokke geneesheer. *Maar hierdie liefdadigheid moet nie misbruik word as 'n maatstaf vir die vasstelling van die maksimum-gelde wat vir private behandeling gevra mag word nie.* Dit is derhalwe van belang om in gedagte te hou dat hierdie spesiale tariewe nooit aktuarieel bereken is nie. Hulle hou geen rekening nie met die vinnige en aansienlike styging in die koste van lewensonderhoud waarteen 'n menigte private burgers in die staatsdiens en in die handel en nywerheid deur die landswette gevrywaar en beskerm word.

Die prys wat die mediese beroep in kontant moet betaal vir die instandhouding van die welsyn van 'n deel van die gemeenskap, word treffend aan die lig gebring deur die geweldige surplusse wat die Kommissaris vir die Skadeloosstelling van Werkliede opgebou het. Ondanks rabatte van letterlik honderdduisende ponde aan ons nywerhede, staan die reserwefonds op sowat £1 miljoen. Hierdie bedrag is in werklikheid oor 'n tydperk van enkele jare deur die dokters van hierdie land uit hul eie sakke bygedra. Die paradoks hier is dat die werkgewer (en nie die pasiënt nie) verantwoordelik vir die betaling van die rekening is. Met ander woorde, die mediese praktisyn subsidieer ook die handel en nywerheid.

Ons vind dit moeilik om te verstaan waarom die Fonds wat deur die Wet op die Skadeloosstelling van Werkliede in die lewe geroep is, in staat behoort te wees om mediese dienste teen 'n verminderde tarief te verkry. Vroeër (en trouens vandag nog) het werkgewers kontrakte met private versekeringsmaatskappye aangegaan, en die dokter het die volle bedrag

fund stands at about £1 million (a sum of money contributed in effect substantially out of the pockets of the doctors concerned in a few years). The paradox here is that the employer (and not the patient) is responsible for the fee. Thus the practitioner subsidizes industry and commerce.

It is difficult to see why the W.C.A. Fund should buy medical services at a reduced rate. Formerly employers contracted (as some, indeed, still do) with private insurance companies and doctors were rightly paid full fees. The Workmen's Compensation Commissioner apparently justifies his demand for a reduced fee by the argument that he buys his medical services wholesale. We have yet to hear of a more fatuous suggestion. No practitioner regards a sick patient as an anonymous item in a purchasing programme. He treats every patient as a human being requiring individual attention. To put it in language the Commissioner can understand, the Commissioner may convince himself he is buying wholesale in quantity: the service he gets is retail in quality.

Clearly the cost of medical care is a social problem geared to the cost of living in general, with the profession always making a double sacrifice, as citizens and as doctors, to help solve it.

The tariff of reduced fees for Medical Aid Societies has evolved over many years through the grace and charity of the profession. At one fell swoop it may be used as evidence that these reduced fees are related to the hypothetical average private fees. It can in that case become a scale against which disputes about private fees may be measured. The tariff, having been stood on its head, will then be forced to play a rôle which was never contemplated for it. It grew up as a special way of helping those in need and was never meant to have any connexion with settling disputes about private fees.

If it becomes a scale applicable equally to everybody in all parts of the country it must operate with occasional injustice both to doctor and patient, because economic factors vary widely in different geographical regions and in different social strata. Medical charges have always varied (and for very good reasons) in different parts of the country, and at different social levels.

That the imposition of a uniform, nation-wide tariff for Medical Aid Societies is in itself wholly unrealistic has been demonstrated in recent months by the decision of the Southern Transvaal Branch of the Medical Association

ontvang. Die Kommissaris grond sy eis om verminderde tariewe blykbaar op die argument dat hy sy mediese dienste op 'n groothandelsgrondslag ‚koop'. 'n Meer onnosele argument as dit kan beswaarlik geopper word. 'n Dokter beskou 'n siek pasiënt nie as 'n anonieme item op 'n aankooplys nie. Hy behandel iedere pasiënt soos 'n mens wat individuele aandag nodig het. Of, om dit te stel in 'n taal wat die Kommissaris kan begryp: Die Kommissaris verkeer miskien onder die indruk dat hy kwantiteitsgewyse—in die groothandel—koop. Die diens wat hy kry is egter kwaliteit—in die kleinhandel.

Dis duidelik dat die koste van mediese behandeling 'n maatskaplike vraagstuk is wat ten nouste aan die algemene koste van lewensonderhoud verwant is. Dis ook duidelik dat die mediese beroep altyd 'n dubbele opoffering moet doen—enersyds as geneeshere en andersyds as gewone burgers—om daardie probleem te help oplos.

Deur die welwillendheid en liefdadigheid van die mediese beroep het die stelsel van verminderde tariewe vir Mediese Hulpverenigings oor 'n tydperk van baie jare tot stand gekom. Nou, eensklaps, kan dit gebruik word om te bewys dat hierdie verminderde tariewe verband hou met die hipotetiese gemiddelde bedrag wat 'n private pasiënt gevra kan word. Dit kan, met ander woorde, die maatstaf word waarmee geskille oor private doktersrekenings gemeet word. Nadat die tarief dan as 't ware onderstebo gedraai en gedwing is om op sy kop te gaan staan, moet dit 'n rol speel wat dit nooit bedoel was om te speel nie. Dit het ontstaan as 'n spesiale manier om hulp aan hulpbehoewendes te verleen, en daar was nooit die minste gedagte dat dit eendag miskien gebruik sou word vir die beslegting van geskille oor private doktersrekenings nie.

As dit 'n skaal word wat van toepassing op almal in alle dele van die land is, kan dit, in bepaalde omstandighede, onbillik teenoor sowel die dokter as die pasiënt wees, want alle aardrykskundige streke en al die verskillende seksies van die samelewing in ons land kan nie oor dieselfde kam geskeer word nie. Doktersrekenings in verskillende dele van die land, en vir die verskillende sosiale stande van die samelewing, het nog altyd gewissel—en daar is 'n baie goeie rede hiervoor.

Dat die oplegging van 'n eenvormige, landswye tarief vir Mediese Hulpverenigings op sigself geheel en al onrealisties is, is gedurende die afgelope paar maande bewys deur die besluit van die Suid-Transvaalse Tak van to give notice of withdrawal from the national schedule laid down; and by the reaction of the Mutual Medical Aid Society (a Society not recognized by the Medical Association) in undertaking to pay fees considerably higher than those scheduled. Moreover, the change in complexion of the former National Medical Aid Society, which now operates as an insurance company, will also materially raise the fees paid by organized groups of citizens to approach the level of normal private fees applicable in various parts of the country.

These events not only reflect a fundamental change in the relationship between the practitioner and those of his patients who organize to make provision for future medical commitments, but also render even more complicated the unenviable duties of the Council in dealing with disputes about private fees on the basis of any arbitrary tariff.

The wisdom of the new arrangement is seriously to be questioned, especially as the Council now finds itself saddled with the commercial function of price control; for the relationship between a doctor and his patient cannot be measured by a tariff set down in print. Indeed, this unique relationship can only be damaged by it.

HISTORY REPEATS ITSELF

It is not out of place to remind ourselves of an earlier attempt at price control of medical fees. In 1894 practitioners in the Orange Free State protested vigorously against the Draft Medical Bill *inter alia* because of a proposed tariff of fees. A leading member of the profession said at the time about the proposed tariff:

'The price charged by any one for any commodity depends upon the value of that commodity, which is regulated by supply and demand. To say, therefore, to a medical man you shall only charge so much for advice or so much for medicine is to attempt to fix what is impossible.

'If an individual practitioner has attained to so much eminence in his profession or has so much repute as to render his services to be in demand, it cannot be attempted to rate his services by a scale which would be no inducement to him to place his skill at the disposal of the community. . . . How is it then, that when it is impossible to fix by tariff the price of . . . horses or pigs, it is attempted to price the brainpower and skill of men who are absolutely necessary to the comfort and well-being of the community. In a Free State governed on republican principles this is a barbarous anomaly which should be left to Kafir potentates . . . but nowadays in South Africa, when freedom has found a home and progress has sounded a new advance, such enactments are dangerous to liberty and are madly erroneous economical fallacies. . . . .

'How has it come to be possible for any com-

die Mediese Vereniging om kennis te gee dat hy hom aan die nasionale bylae, soos vasgestel, gaan onttrek, en deur die reaksie van die Onderlinge Mediese Hulpvereniging ('n vereniging nat nie deur die Mediese Vereniging erken word nie) wat onderneem het om gelde, veel hoër as dié wat in die bylae verskyn, te betaal. Temeer, die veranderde houding van die voormalige Nasionale Mediese Hulpvereniging, wat tans as 'n assuransiemaatskappy optree, sal ook daadwerklik bydra tot 'n verhoging van die gelde wat deur georganiseerde groepe burgers betaal word, en sal hierdie gelde nader bring aan die peil van die normale private doktersgelde wat in verskillende dele van die land betaalbaar is.

Hierdie voorvalle weerspieël nie alleen 'n fundamentele verandering in die verhouding tussen die praktisyn en sy pasiënte wat hulle in groepe georganiseer het sodat hulle voorsiening vir toekomstige mediese verpligtings kan maak nie, maar bemoeilik ook die reeds onbenydenswaardige taak van die Raad as die Raad geskille oor private gelde op grondslag van 'n arbitrêre tarief moet besleg.

Oor die wysheid van die nuwe reëling bestaan daar dus ernstige twyfel, veral noudat die Raad belas is met 'n kommersiële funksie, nl. prysbeheer. Want die verhoudinge tussen 'n dokter en sy pasiënt kan nooit gemeet word aan die hand van 'n gedrukte tarief nie. Trouens, so 'n tarief kan daardie unieke verhouding net beskadig.

DIE GESKIEDENIS HERHAAL HOMSELF

Dit is nie onvanpas nie om ons hier te herinner aan die poging wat vroeër aangewend is om prysbeheer op doktersrekenings toe te pas. In 1894 het mediese praktisyns in die Oranje-Vrystaat hewige protes teen die Ontwerpwet op Geneeshere aangeteken, onder meer omdat dit voorsiening gemaak het vir 'n tarief van doktersgelde. 'n Vooraanstande lid van die beroep het hom destyds soos volg oor die voorgestelde tarief uitgelaat:

„Die prys wat deur enigeen van ons vir enige artikel gevra word, hang af van die waarde van daardie artikel. Dit, op sy beurt, word weer gereël deur vraag en aanbod. Om derhalwe aan 'n mediese praktisyn te sê: Jy moet slegs soveel vir jou advies vra, of soveel vir jou medisyne, is niks anders nie as 'n poging om die onmoontlike vas te stel.

„As 'n individuele praktisyn so 'n hoogstaande posisie in sy beroep verower het, of so 'n goeie reputasie geniet dat daar 'n aanvraag om sy dienste bestaan, is dit onmoontlik om daardie dienste te waardeer ooreenkomstig 'n skaal wat geen aansporing vir hom sal wees om sy vernuf tot beskikking van die gemeenskap te stel nie . . . as dit onmoontlik is

munity to fancy it can increase the efficiency and character of a highly honourable and deserving profession by a repressive legislation is difficult to understand. In older countries for ages and ages the profession of medicine has been regarded as of the highest honour, and every effort has been made to stimulate the development of that profession, honours have been showered on its professors, rich emoluments have been placed at their disposal, wise laws have been enacted for its guidance, but nowhere in the wide world, except in South Africa, has any attempt ever been made to fix by law the price of medical advice and medicines. Again something new from Africa!'[1]

The insinuation of this undesirable commercial feature into medical practice was successfully prevented in the nineteenth century. Now some 60 years later, we find ourselves the victims of just such a system, and there is current a lamentable tendency to decry well-merited attempts to rectify the deplorable economic situation threatening so many medical practitioners to-day.

The humanistic tradition of our profession, throughout its long and honourable history, has been to contribute to the advancement of medical science, as well as to the social, cultural and academic developments of the day. The profession can only fulfil these functions in an environment in which every individual practitioner enjoys a reasonable prospect of achieving economic security. But the extent to which these altruistic aspirations can be attained is in direct relation to the economic status of the profession itself.

The time has therefore clearly come when all concerned should re-apply themselves to a rational assessment of the issues involved, as our medical forebears so pertinently did in 1894.

* * * *

The same things are not proper for all . . . a casual trial may perform that which a rational process cannot. *Celsus.*

THE WISDOM OF HIPPOCRATES

It is impossible to cure a severe attack of apoplexy and no easy matter to cure a mild one.

THE STATUS OF THE MEDICAL PROFESSION

'. . . if our present trend continues another result must surely be not only the extinction of the well-educated man but the down-grading of our profession in the eyes of others to the level of a skilled trade, with results to be felt personally and practically by all its members. It is hardly fanciful to suppose that in the minds of some of the engineers

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1. S. Afr. Med. J., May 1894, p. 27.

om die prys van . . . perde of varke volgens 'n bepaalde tarief vas te stel, hoe is dit dan moontlik dat 'n poging aangewend kan word om die prys van die denkvermoë en behendigheid van manne wat absoluut onontbeerlik vir die gerief en welsyn van die gemeenskap is, vas te stel? In 'n Vrystaat wat volgens republikeinse beginsels geregeer word, is dit 'n barbaarse anomalie wat ons liewer aan die Kafferkonings moet oorlaat . . . in Suid-Afrika waar vryheid 'n tuiste gevind het en vooruitgang aan die orde van die dag is, hou so 'n wetsbepaling 'n gevaar vir vryheid in, en is dit 'n malle, ekonomiese drogredenasie . . .

„Hoe dit vir enige gemeenskap moontlik is om hom vir 'n enkele oomblik te verbeel dat hy die doeltreffendheid en karakter van 'n eerbare en verdienstelike beroep kan verbeter deur onderdrukkende wetgewing, val moeilik om te begryp. In die ouer lande word die mediese beroep al eeue en eeue lank as hoogs eerbaar beskou, en alle moontlike pogings is in die werk gestel om die ontwikkeling van daardie beroep te bevorder. Sy professore is met eerbewyse oorlaai, hoë salarisse is aan hulle betaal, en verstandige wette is aangeneem om as leidraad vir hulle te dien. Maar nêrens in die wêreld, met uitsondering van Suid-Afrika, is 'n poging aangewend om die prys van mediese raad en medisyne kragtens wet vas te stel nie. Weereens kom daar dus iets nuuts uit Africa.' [1]

Die insypeling van hierdie onwenslike kommersiële kenmerk in die mediese praktyk is met welslae in die negentiende eeu bestry.

Nou, sowat 60 jaar later, vind ons dat ons die slagoffers geword het van presies so 'n stelsel, en 'n betreurenswaardige neiging tot kleinering van die verdienstelike pogings wat aangewend word tot verbetering van die uiters ongemaklike ekonomiese posisie waarin so baie mediese praktisyns hulle vandag bevind, is reeds bespeurbaar.

Dwarsdeur sy lang en eerbare geskiedenis was dit die humanistiese tradisie van ons professie om 'n bydrae te lewer tot die bevordering van die mediese wetenskap sowel as tot die sosiale, kulturele en akademiese ontwikkeling van die dag. Die professie kan hierdie funksies alleen nakom in 'n omgewing waar iedere individuele praktisyn 'n redelike vooruitsig op ekonomiese veiligheid geniet. Die mate waarin hierdie altruïstiese oogmerke bereik kan word, staan trouens in regstreekse verhouding tot die ekonomiese status van die professie self.

Die tyd het derhalwe aangebreek wanneer alle belanghebbendes hulle opnuut moet toewy aan 'n rasionele benadering van die vraagstukke wat by die saak betrokke is, net soos ons mediese voorgangers so pertinent in die jaar 1894 gedoen het.

1. S. Afr. Med. J.. Mei 1894, bl. 27.

and draughtsmen of the National Health Service we were already regarded not as a profession to be consulted but as so much skilled labour to be engaged when the scheme was sufficiently advanced'.

(Lindsey W. Batten, M.B., M.R.C.P. on *Adult Medical Education*: *A General Practitioner's Viewpoint* in *Brit. Med. J.*, 27 August 1955).

THE DUTIE OF A CHIRURGIAN

Five things are proper to the dutie of a Chirurgian:
To take away that which is superfluous,
To restore to those places, such things as are displaced,
To separate those things which are joyned together,
To joyn those which are separated,
To supply the defects of Nature.

Thou shalt fare more easily and happily attaine to the knowledge of these things by long use and much exercise, than by much reading of Bookes, or daily hearing of Teachers. For speech, how perspicuous and elegant soever it be, cannot so vively expresse any thing, as that which is subjected to the faithfull eyes and hands . . . neither may we doubt but that studies would at the length waxe cold, if they, only furnished with the Theoricke and Precepts in Schooles and that with much laboure, should see no manuall operation, nor manifest way of performing the Arte.

(From *The Epistle Dedicatorie—To Henry the Third, the most Christian King of France and Poland.* Ambroise Paré. Paris. 8 February. Anno Domini 1579.)

CORTISONE-INDUCED PSYCHOSIS

Our readers will be interested in an instructive account of a cortisone-induced case of manic-depressive psychosis which appeared under the title *Annals of Medicine* in the issue of *The New Yorker* dated 10 September 1955, at p. 47.

The author is the well-known writer Berton Roueché, whose record of the insidious development of euphoria, as well as of bulimia and alternating manic and depressive states is clinically very revealing.

The patient was being treated for peri-arteritis nodosa.

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HYPERTENSION AND ITS TREATMENT

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Hypertension has been said to be the greatest killer of civilized peoples. It is certainly a very common cause of disablement and is encountered by practitioners of almost every branch of medicine. Despite this, hypertension remains in all aspects a controversial condition.

AETIOLOGY

Of the aetiology of hypertension and especially of the variety known as essential hypertension, little is known. Certain facts are however becoming clearer. The role of inheritance is now established.[1, 2] It is said that if both parents are hypertensive, there is a 45% incidence of hypertension in the offspring; whereas if neither parent is hypertensive there is only a 3% incidence.[2] This fact is important in assessing the prognosis in individual patients.

In essential hypertension there is a more or less generalized vasoconstriction, but whether this vasoconstriction is due to vasoconstrictor substances or neurogenic influences is not yet known.[1] Pressor substances in the blood, such as pherentasin,[3] V.E.M. (Vaso-Excitor Material),[4] from anoxic tissue (especially kidney) and in particular hypertensin or angiotonin[5] formed by the release of renin from ischaemic kidney to join hypertensinogen in the circulating blood, have all been invoked as causes of hypertension.

Endocrine influences are also no doubt of considerable importance. The secretion of adrenaline and noradrenaline by the phaeochromocytoma of the adrenal medulla is responsible for the paroxysmal and sometimes persistent hypertension that occurs with these tumours. Less certain but more intriguing is the influence of the adrenal cortex and its relationship to sodium metabolism in the aetiology of various forms of hypertension. Certainly in severe adrenal cortical depletion, hypertension is an impossibility.

For practical treatment a broad classification of the causes of hypertension is essential Table 1). It will be noted that 80% of hypertensives fall into the category of essential and malignant hypertension.

TABLE 1: CLASSIFICATION OF HYPERTENSION

A. Essential Hypertension B. Malignant Hypertension	} 80%
C. Renal Hypertension.	
e.g. Chronic glomerulo-nephritis.	
Pyelo-nephritis.	
Polycystic kidneys.	
D. Unilateral Renal Disease.	
E. Other Causes:	
Phaeochromocytoma.	
Cushing's syndrome.	
Coarctation of the aorta.	
Toxaemia of pregnancy.	

THE NORMAL BLOOD PRESSURE

Since Janeway[6] using Riva-Rocci's sphygnomanometer, reported on clinical blood pressure recordings in 1903, we should by now know what are the normal pressure limits, but even these basic facts are clouded in uncertainty.

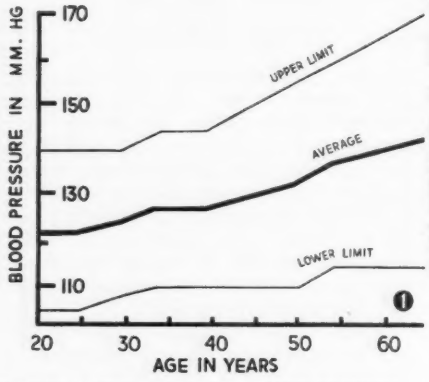
It was first said that 100 + age represented the normal systolic pressure in mm. Hg, but later a normal pressure was taken to be 120 mm. Hg systolic and 80 mm. Hg diastolic, with an upper limit of 140/90 mm. Hg. To-day the work of Pickering[1] in England and Master and his colleagues in the United States[7] has made it clear that both diastolic and systolic pressures increase with age. Figs. 1—4 show the average systolic and diastolic pressures at various ages and are taken from Master's data.[7] It is obvious that views on what constitutes a normal pressure require drastic revision in order to accord with these figures.

NEW CONCEPTS OF HYPERTENSION

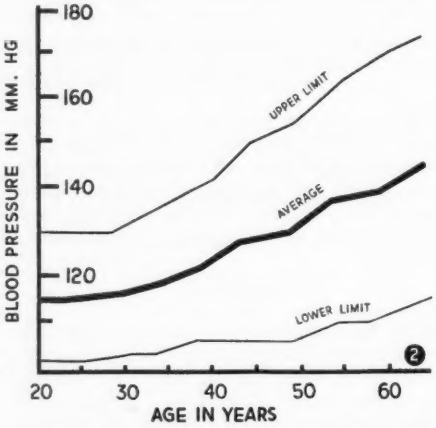
A concept of importance is the fact that the systolic pressure is of little significance and that the severity of the hypertension is determined by the height of the diastolic pressure. A raised systolic pressure is normal in the elderly person with an inelastic aorta and also occurs in bradycardia, aortic insufficiency, beriberi, anaemia and in response to exercise or emotion. In such conditions the term *systolic hypertension* would be accurate. The elderly individual with a blood pressure of 180/100 mm. Hg is, in reality, not a hypertensive but an arteriosclerotic subject.

It would be better if genuine hypertension were referred to as *diastolic hypertension*.

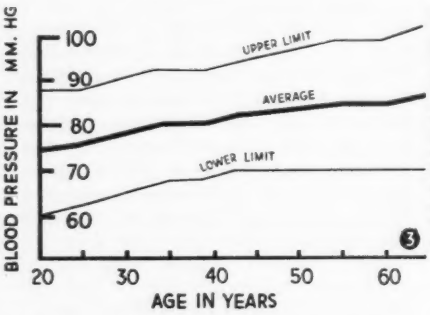
NORMAL RANGE—SYSTOLIC BLOOD PRESSURE (MALES)



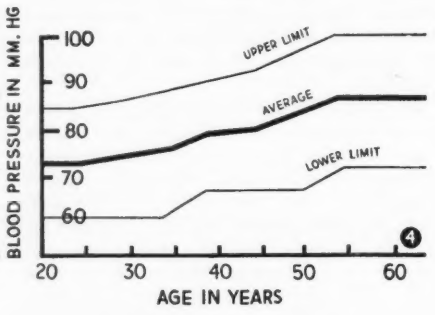
NORMAL RANGE—SYSTOLIC BLOOD PRESSURE (FEMALES)



NORMAL RANGE—DIASTOLIC BLOOD PRESSURE (MALES)



NORMAL RANGE—DIASTOLIC BLOOD PRESSURE (FEMALES)



Two other important concepts which are not generally recognized are those of the 'hyper-reactor' and of 'labile hypertension'. These two concepts are actually related and are concerned with the difference between 'basal' and 'casual' blood pressures.[8]. Basal blood pressure is recorded in the morning after the person awakes but before he gets out of bed, while casual blood pressure is obtained at any other time of the day during ordinary activities. Every physician is familiar with the patient who when first seen has a blood pressure of 160/100 mm. Hg and at a subsequent visit one of 130/80 mm. Hg. Such a patient has a labile hypertension and 'hyper-reacts'. There is no doubt that the systolic pressure may be raised 50 mm. or more, and the diastolic 10—20 mm. by emotion or exertion. However, extreme lability, especially in a young person, must not be disregarded, for out of this group a small percentage, variously estimated at from 3—18%, will develop a 'fixed', 'established' or 'continued' hypertension.

SYMPTOMS AND SIGNS OF HYPERTENSION

There is not even unanimity about the symptoms of hypertension. It would appear that uncomplicated hypertension has *no* symptoms, and that symptoms are the results of complications, i.e. of 'hypertensive vascular disease'. The 2 symptoms most commonly ascribed to hypertension are headache and giddiness, which are the most common symptoms of anxiety.

In regard to the one symptoms of headache I have analyzed 50 consecutive cases of established hypertension from my personal records, and 50 consecutive records of patients with diastolic pressure of 80 mm. or lower at consulting room visits. In Fig. 5 the presence

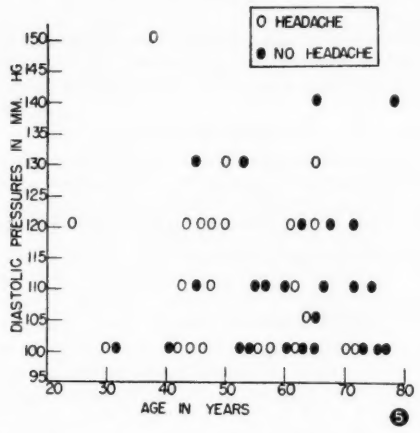


Fig. 5. Headache in 50 consecutive hypertensive patients.

or absence of headache is charted against the age on the abscissa and the diastolic pressure on the ordinate. It can be seen that headache or its absence bears no constant relationship to either of these factors and, in fact, some patients with the highest diastolic pressure are free from headache.

The relationship between the presence and absence of headache and the severity of the hypertension was also investigated. No significant difference between the 2 groups was revealed (Table 2). Headache occurred in 50% of the cases. The situation of the headache in relation to the level of the diastolic pressure in the 50 hypertensive patients and the 50 normotensive patients was also analyzed (Table 3). From this it would appear that headache is perhaps more common and more often occipital in the hypertensive group, but that neither headache nor its situation are diagnostic of hypertension or its severity.

TABLE 2: RELATION BETWEEN PRESENCE OF HEADACHE AND SEVERITY OF HYPERTENSION

	No. of Patients	*Average Diastolic Pressure*	*Left Ventricular Hypertrophy (On E.C.G.)*	*Fundi Grade 2 or more*	*Left Ventricular enlargement (On X-ray)*
Headache	25	113	15	4	15
No headache	25	111.2	14	4	14

TABLE 3: HEADACHE IN RELATION TO THE DIASTOLIC BLOOD PRESSURE

No. of Cases	*Diastolic Pressure (in Mm. Hg.)*	*Headache*			
		Occiput	*Vertex*	*Others*	*Nil*
18	120+	9	1	3	7
13	110—119	3	0	3	8
19	100—109	7	1	2	10
Total= 50 F=26, M=24		19	2	8	25
Total= 50 F=25; M=25	80—	8	3	8	32

Diastolic over 100 Mm. Hg.—26 females, 24 males.
Diastolic under 80 Mm. Hg.—Sexes equally divided.

These findings are in keeping with those of other observers.[9]

The signs of hypertension apart from the blood pressure itself are those due to hypertensive vascular disease and cerebro-vascular disease.

INVESTIGATION OF PATIENTS

Severe hypertension requires investigation in order to establish the prognosis and, if possible, the aetiology for purposes of treatment. The minimal investigations required are shown in Table 4. It is possible that the inclusion of the simple Regitine test as a routine might help to discover more tumours of the phaeochromocytoma type. Regitine blocks the effect of adrenaline and noradrenaline on the sympathetic vaso-constrictor fibres, thus causing a significant drop in pressure in cases of tumours secreting these substances. It is simpler and safer to use than piperoxane, but possibly less accurate.[10] The estimation of urinary amines during hypertensive phases is also a most useful procedure in the diagnosis of adrenal medullary tumours.

TABLE 4: MINIMAL INVESTIGATIONS IN SEVERE HYPERTENSION

A. Heart: X-ray, E.C.G.
B. Ophthalmoscopic Examination.
C. Renal Function.
(*a*) Urine.
(*b*) Concentration test.
(*c*) Blood urea.
(*d*) I.V.P.
D. Benzodioxane or Regitine Test.

To this list of tests one might with advantage add the sedation test with amytal or nembutal in order to discover the basal pressure. Frequently patients are seen with a constant diastolic pressure of 120 to 130 mm. Hg on casual readings in the Out-Patient Department, whereas a sedation test might show a basal diastolic pressure of 90 mm. Hg. These are the patients who, if observed in a hospital ward or treated by psychotherapy, will attain normal or near normal pressures and the improvement will be credited to some placebo. It has been shown that repeated blood pressure readings in any single patient will usually show some decrease over the first 5 or 6 weekly readings or even a continuous decrease over a period of months, without any treatment.[11, 12]

PROGNOSIS

The severity and prognosis of any individual case of hypertension can be assessed by studying 5 factors (Table 5). These factors should be considered in relationship to age, sex and family history. Younger persons in general have a worse prognosis, while females are known to stand hypertension particularly well. The retinal changes (Table 6) are probably the most important single prognostic indicator.[13, 14] Assessment of severity will assist in deciding on the type of treatment.

TABLE 5: PROGNOSIS

A. Established hypertension with complications: 80 per cent. fatal in 10 years.
B. Established hypertension without complications: 20 per cent. fatal in 10 years.
Judge severity on changes in:
1. Retina.
2. Heart.
3. Kidneys.
4. Brain.
5. Diastolic Pressure.
6. Background of Age and Sex.

TABLE 6: FROM WRIGHT[14], MODIFIED FROM KEITH[13] *et al.*

1.	Narrowing or sclerosis of retinal arteries.	70% alive after 7 years.
2.	Moderate sclerosis and perhaps some white hard patches.	50% alive after 5 years.
3.	"Cotton wool" exudates, haemorrhages and oedema of retina + above vascular changes.	75% dead in 3 years.
4.	Above changes + papill. oedema.	90% dead in 1½ years.

TREATMENT

If there is an aetiological factor such as coarctation of the aorta (and if the femoral arteries are palpated as a routine, this diagnosis is not difficult), a phaeochromocytoma or a severe unilateral renal lesion is found, these must be dealt with surgically.

We are concerned here mainly with essential hypertension or hypertension due to bilateral renal disease.

For treatment purposes there are 4 groups:

(*a*) *Malignant hypertension,* which all authorities agree requires immediate treatment.

(*b*) *Established hypertension with complications* in people under the age of 70 years requires treatment. This applies especially to younger patients with early cardiac failure, a history of cerebro-vascular accidents or retinal changes.

(*c*) *Established hypertension with a diastolic pressure of* 110 *to* 130 *mm. Hg without symptoms,* especially if a sedation test shows a high basal pressure, probably requires treatment, or at the least twice yearly observation for deterioration. This is the controversial group, but I would suggest prophylactic treatment in these patients in view of the dangers they are exposed to. It is possible that females could be watched, but in view of the greater risks run by males, the latter should be treated.

(*d*) *Labile hypertension* presents many difficulties. These patients can also have cerebro-vascular or electrocardiographic and other changes; if so, they require treatment. The difficulty arises in the cases with no objective signs and occasional normal pressure readings. These probably require only reassurance and common sense psychotherapy together with mild sedation or the use of Rauwolfia.

There are now available many methods of lowering blood pressure, but only the important ones will receive consideration here.

1. *Psychotherapy.* Psychotherapy is probably required in all cases of hypertension, but has its main value in the labile or mild hypertensive. Simple explanation, reassurance and instruction on how to avoid stress-provoking situations are all that is required.

2. *Diet.* Dietary measures have long been used in treatment. Weight reduction has been practised assiduously in the past, but there is no real evidence of its efficacy.[11, 15, 16] However, weight reduction confers other benefits such as diminished work load for the heart, despite its failure to lower significantly the diastolic pressure.

Salt restriction is mainly of value in actual or impending cardiac failure. Even a drastic restriction of sodium to under 200 mg. daily only produces a significant decrease in blood pressure in about 20% of cases.[17, 18] The usual 'salt poor' rather than a true 'low salt' diet has no therapeutic effect on the blood pressure. Drastic salt restriction is contra-indicated in the presence of advanced renal failure, and may precipitate weakness, nausea and azotaemia.[17]

The 'rice diet' exerts its effects by virtue of its low sodium content, but its low protein content makes it valuable in cases of uraemia. The unpalatability of these diets renders them impractical for prolonged use.

3. *Chemotherapy.* A number of potent hypotensive drugs is now available. Some such as Hydergine, potassium thiocyanate and sodium nitroprusside have fallen into disuse, but the following 4 groups of drugs are becoming increasingly popular.

(*a*) *Rauwolfia serpentina,* an Indian herb, is available in 3 main forms (Table 7). There is no proof that there is any essential difference in the action or side effects of the 3 forms.[19, 20] It is said that Reserpine has a more sedative effect than either the Alseroxylon fraction or the crude root, but the difference appears to be negligible.

TABLE 7: RAUWOLFIA SERPENTINA

Preparation	*Trade Name*	*Tablet Size (In mg.)*	*Daily Dose (In mg.)*
Crude Root	Raudixin	50 and 100	200–1,000
Alkaloidal Extract	Rauwiloid	2	4–16
Single Alkaloid (Reserpine)	Serpasil Serpiloid	0.1 and 0.25	0.5–3.0

Rauwolfia probably exerts its main effects on the hypothalamus. Sympathetic inhibition is said to relax vasoconstrictor tone, while parasympathetic stimulation causes bradycardia and increased bowel action[20] (Table 8). Among their side effects they produce tranquillity without somnolence and in this respect these drugs are superior to the barbiturates. In some patients, however, they cause depression, which restricts their use. Nasal stuffiness, muscular pains and weight gain may also occur during their use (Table 8).

TABLE 8: RAUWOLFIA

Action: 1. Sympathetic inhibition.
2. Parasympathetic stimulation:
Bradycardia.
Bowel motility.
(These probably occur at the hypothalamic level).
Side Effects: Nasal stuffiness, Sedation (tranquillity), Increased bowel movement. Dreams, Muscular pain, Increase in appetite and weight.

The effect of Rauwolfia is usually noticeable in 3—6 days, but may take 3—6 weeks to appear and may persist for 3—6 weeks after discontinuation of the drug.[17, 18]

These drugs are only mildly hypotensive and find their main use in mild or labile hypertension, or as adjuvants to other drugs such as hydralazine and the methonium compounds. They overcome the constipation due to drugs such as pentolinium (Ansolysen) and the tachycardia caused by hydralazine.

In view of the fact that they do not cause postural hypotension, there are practically no contra-indications to their use.

Dosage is an individual problem, but should be big enough to produce minor degrees of nasal stuffiness and bradycardia. Average doses are shown in Table 7.

(*b*) *Veratrum viride* derivatives are perhaps less useful because of the narrow margin between therapeutic and toxic effects. They exert vaso-depressor and bradycardic effects at the hypothalamic level, and also act on pressor receptors in the coronary arteries and carotid sinus.[20] In ordinary doses they cause no postural hypotension, but with doses only slightly higher than suitable therapeutic doses, they cause substernal and epigastric burning, vomiting, shivering and collapse (Table 9). The

TABLE 9: VERATRUM

Action:	1. Vasodepressor and bradycardiac at the hypothalamic level. 2. Von Bezold or "coronary chemoreflex". 3. Effect on carotid sinus.
Side Effects:	Bradycardia, Substernal and epigastric "burning", Nausea and vomiting, Collapse.

TABLE 10: VERATRUM PREPARATIONS AND DOSES

Crude Drugs: Veriloid and Vergitryl.	1 or 2 tablets 4 times daily *after meals.*
Protoveratrine A + B (Provell Maleate, Puroverine).	0.75 to 1.5 mg. twice daily after meals with a booster of 0.25 mg. 2 hours later.

available products and suitable doses are shown in Table 10.

It was hoped that the purified product protoveratrine might be more useful, but this drug appears to have the same disadvantage as the other Veratrum preparations.[20] For hypertensive crises or eclampsia protoveratrine (Puroverine) can be given slowly intravenously in doses of 0.1—0.2 mg. If used in combination with digitalis, Veratrum may, because of its bradycardic effect, product heart block, but this can be relieved by the use of atropine.[17]

(*c*) *Hydralazine* (Apresoline, Table 11). Hydralazine probably produces its main effect

TABLE 11: HYDRALAZINE (APRESOLINE)

Action:	1. Direct vasodilator action. 2. Central action on hypothalamus. 3. Inactivation of pressor substances.
Side Effects:	Headache, tachycardia, dyspnoea, paraesthesiae, nervousness, dry mouth, anxiety, depression, fever, arthritis, L. E. syndrome.
Dose:	25—200 mg. 4 times daily.

by a direct vasodilator action on the arterioles[21] as well as by a central action on the hypothalamus and possibly by inactivation of pressor substances such as pherentasin.[3] It has been said to cause an increased renal plasma flow,[21] but there is no increase in glomerular filtration rate. The increased renal plasma flow may improve the nutrition of the renal tubular cells.[3, 18] Its hypotensive effect is only moderate and its action is delayed for days to weeks.[20] In view of its side effects it is advisable to restrict its use to combinations with other antihypertensive drugs such as Rauwolfia. It sometimes produces angina or just a substernal burning feeling, even without producing a lowering of blood pressure. In large doses it may produce pyrexia, arthritis and a syndrome resembling disseminated lupus erythematosis. In view of this the daily dose should be kept under 300 mg.

(*d*) *Ganglion Blocking Compounds.* These are undoubtedly the most effective of the hypotensive agents. They produce their effects by blockade of sympathetic ganglia but also affect parasympathetic ganglia. This latter action is mainly responsible for side effects, such as impairment of gastric and bowel motility; paralytic ileus has been known to occur. Other unpleasant side effects are difficulty in accommodation causing blurred vision, urinary retention, dry mouth and anorexia, impotence and, most important of all, postural hypotension with syncope.

The products available here are methonium compounds such as hexamethonium bromide or bitartrate (Vegolysen) and hexamethonium chloride (Methium) but these have largely been supplanted by pentolinium tartrate (Ansolysen) because of the latter's more prolonged action and more consistent effects by the oral route.

In severe hypertension, Smirk advises initial small doses such as 3 mg. 12-hourly of subcutaneous pentolinium (Ansolysen) administered with a tuberculin syringe.[22] At the outset the pressure is measured in the erect posture every ½ hour during the day. The ideal result is a pressure of 120/80 mm. Hg

TABLE 12: DOSE AND INCREMENT OF METHONIUM COMPOUNDS

Product	*Initial Dose (Mg.)*	*Increments (Mg.)*	*Final Total Daily Dose (Mg.)*	*Average Duration of Significant Action*
Ansolysen Oral	20	20	1400	8—12 hours+
Ansolysen Retard (Subcutaneous Injection)	3	0.5—1.5	140	8—12 hours+
Vegolysen Bromide (Aqueous Injection)	15	5	1200	2—3 hours

in the 'trough period', i.e. the period of maximum activity. The usual dose and increments advised by Smirk are shown in Table 12.

Postural hypotension and giddiness due to these drugs is due to a decrease in cardiac output, a loss of venomotor tone in the legs, and a splanchnic pooling of blood in the erect posture (Fig. 6). These effects are reversed by lying down, walking or abdominal pressure.

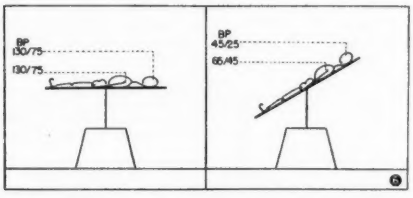


Fig. 6. The effect of posture on blood pressure in a patient on hexamethonium bromide. (From Prof. W. D. M. Paton's contribution on *The Principles of Ganglionic Block*) in *Lectures on the Scientific Basis of Medicine* (Vol. 2, p. 156), published by The Athlone Press, London. By permission of the author and the publishers.

It is usual to start with an oral dose of 20 mg. Ansolysen twice daily, and if it is impossible to take blood pressure readings half hourly, the dose can be adjusted by means of the 'one minute test'. The patient is instructed to stand quite still for one minute before each dose. Fainting implies too big a dose; one should aim at slight giddiness. A dose of half the morning dose should be taken at midday, and the one minute test can be applied half hourly between doses in order to find the period of maximum activity and also the correct individual dosage.

The patient should be instructed to take the tablets *with a glass of water, a half hour before meals.*

Table 12 shows the dosage and daily increments suggested by Smirk.[2] In view of the fact that the hypotensive effect is greater in the erect position, patients should sleep with the head of the bed blocked, and should be instructed to keep in a sitting or standing position for as long as possible during the day.

The critical dosage of the drug and its inherent dangers make it a measure which is usually beyond home care. Constipation with irregular absorbtion of the drug and increased tolerance over the first few weeks, require unceasing vigilance.

(*e*) *Combinations of Drugs.* These are now the most popular form of treatment in hypertension. The most effective combination appears to be a Rauwolfia product with pentolinium but oral hexamethonium with Hydralazine (Hyphex[24]), and 'cocktails' consisting of 3 or more hypotensive drugs plus prostigmine to overcome constipation are also used.

There is no doubt that these medical measures prolong life and reverse retinal and cardiac changes in severe hypertension.

4. *Surgery.* Surgical treatment of hypertension remains controversial. In the young patient who is doomed to a life of perpetual medication, lumbo-dorsal sympathectomy is of undoubted value. While it is true that approximately one third of the patients get no response, one third do get a good symptomatic and objective response and one third will get a dramatic response. Even those who get no response are usually more responsive to the effects of hypotensive drugs than they were before operation. The *modus operandi* of sympathectomy is still questionable, but presumably release of sympathetic tone and postural pooling of blood in the extremities play some part. The operative mortality is small—under 2% in good hands—

and after the initial post-operative stage, the patient's co-operation is no longer required.

The latter is an important consideration in dealing with chronic disease in young individuals. Furthermore the results when good are not attended with the unpleasant side effects of drugs, which Paton has detailed so graphically in his description of the 'hexamethonium man'.[23] To quote Paton, 'the "hexamethonium man" is a pink-complexioned individual except when he has stood in a queue for some time, when he may get pale and faint. His handshake is warm and dry. He is a placid and relaxed companion; he may laugh, but he cannot cry because the tears cannot come. Your rudest story will not make him blush and the most unpleasant circumstances will fail to make him turn pale. His wife will testify that his collars and socks stay very clean and sweet. He wears corsets and may, if you meet him out, be rather fidgety (the corsets compress his splanchnic vascular pool, the fidgets keep the venous return going from his legs). He dislikes speaking much unless helped with something to moisten his dry mouth and throat. He is rather long-sighted and easily blinded by bright light. The redness of his eyeballs may suggest irregular habits and in fact his head is rather weak. But he always behaves like a gentleman and never belches or hiccups. He tends to get cold and keeps well wrapped up. But his health is good; he does not have chilblains and those diseases of modern civilization, hypertension and peptic ulcer pass him by. He is thin because his appetite is modest; he never feels hunger pains and his stomach never rumbles. He gets rather constipated so that his intake of liquid paraffin is high. As old age comes on, frequency, precipitancy and strangury will not worry him, but he will suffer from retention of urine and impotence. One is uncertain how he will end, but perhaps if he is not careful, by eating less and less and getting colder and colder, he will sink into a symptomless hypoglycaemic coma and (like the universe) die a sort of entropy death.'

The present writer can think of at least one patient with malignant hypertension who has lived considerably more than the few months prognosticated before she was treated with reserpine and pentolinium, but who has been made completely miserable by the blurred vision due to difficulty in accommodation, the dry mouth and consequent anorexia, and the continual need for strong purgatives.

Bilateral subtotal or total adrenalectomy has recently been performed in cases of malignant hypertension. These patients are subsequently maintained on small doses of cortisone. It is too soon to evalute the results of this operation, but it would appear to be indicated in the patient who has become entirely resistant to all drugs or who is not prepared to co-operate or persist with drug treatment.

This brief review, while considering certain aspects of hypertension and its treatment, will it is hoped make obvious the optimism with which to-day's practitioner may guide the hypertensive patient, in sharp contrast to the sense of frustration he felt only one decade ago.

OPSOMMING

Sekere aspekte van hipertensie en die behandeling daarvan word in oënskou geneem.

Hieruit blyk duidelik waarom die mediese praktisyn die behandeling van hipertensie kan onderneem met 'n optimisme wat in skerp teenstelling staan met die gevoel van verydeling waarmee hy dieselfde probleem slegs ongeveer 10 jaar gelede aangepak het.

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PRINCIPLES OF UNIPOLAR ELECTROCARDIOGRAPHY

AN INTRODUCTION

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This account does not aim to be a complete or comprehensive treatise on electrocardiography, nor does it seek to supplant the major works on the subject. It is a stepping-stone to the fuller and more detailed study of a most important branch of medical science.

Those introduced for the first time to the intricacies of electrocardiography, are frequently bewildered and sometimes overwhelmed by complicated methods of presentation. The primary object has been to give the beginner a working knowledge of the subject. Consequently, theoretical considerations have been reduced to a minimum, emphasis being placed on practical aspects.

I. BASIC PRINCIPLES

ELECTRICAL ACTIVITY OF THE VENTRICLES

When an electrical impulse flows towards a unipolar electrode the galvanometer will record a positive or upward deflection (Fig. 1).

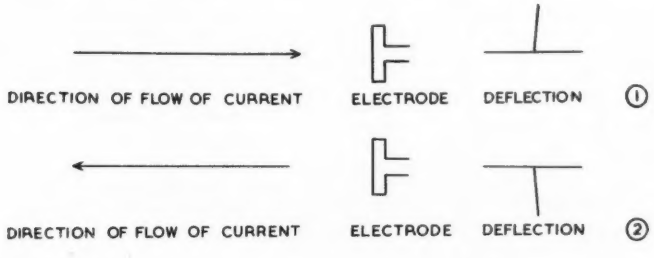
When the impulse flows away from an electrode the galvanometer will record a negative or downward deflection (Fig. 2).

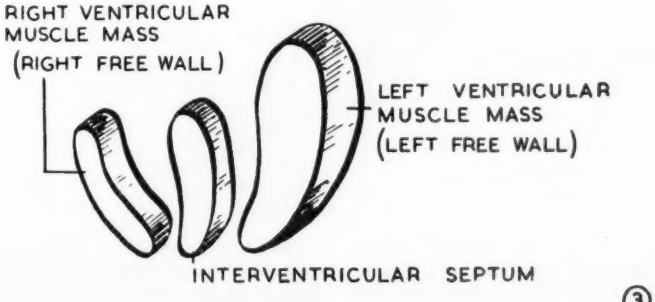
This electrical impulse is termed the depolarization wave. The electrical activity so initiated is termed depolarization.

DEPOLARIZATION OF THE VENTRICLES

Electrocardiographically the ventricles are composed of 3 muscle groups: the interventricular septum, the free wall of the right ventricle (the right ventricular muscle mass) the free wall of the left ventricle (the left ventricular muscle mass) (Fig. 3).

Depolarization commences in the left side of the interventricular septum (Fig. 4, arrow) and spreads through the septum from left to





right. This is the first stage of depolarization (indicated by 1 in Fig. 4).

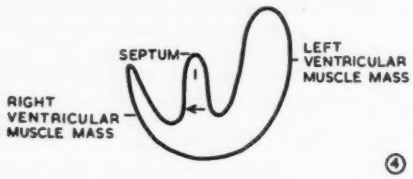


Fig. 4. First stage of depolarization.

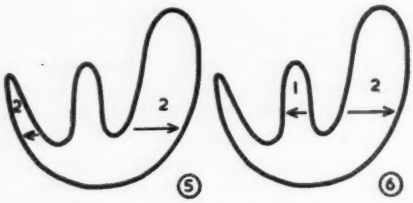
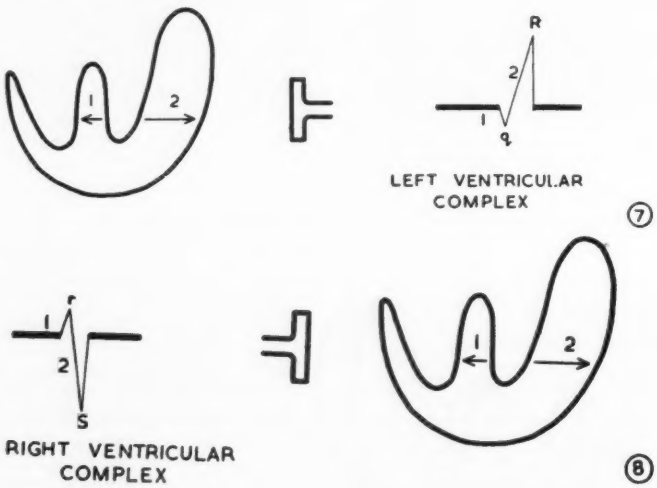


Fig. 5. Second stage of depolarization.
Fig. 6. Depolarization of the ventricles in a simplified form.

Depolarization then proceeds outwards (Fig. 5) simultaneously through the free walls of both ventricles from endocardial to epicardial surfaces. This is the second stage of depolarization (indicated by 2 in Fig. 5).



The free wall of the left ventricle has a larger muscle mass (and hence a larger potential electrical force) than that of the right free wall (Fig. 6). Consequently, as depolarization of both free walls occurs simultaneously, the larger left ventricular forces normally counteract and in effect nullify the smaller forces of the right ventricle. Thus, for convenience, it may be considered that in effect right ventricular depolarization is not normally recorded on the ECG. In a simplified form, depolarization of the ventricles may be shown as a small initial force from left to right across the septum (Fig. 6) followed by a large force from right to left through the free wall of the left ventricle.

An electrode facing the left ventricle will therefore show an initial small downward deflection (called the q wave) caused by the spread of the stimulus *away* from the electrode through the septum, followed by a larger upward deflection (or R wave) caused by the spread of the stimulus *towards* the electrode through the left ventricular mass (Fig. 7). The result is a qR or left ventricular complex.

Conversely, an electrode facing the right ventricle (Fig. 8) will show an initial small upright deflection (called the R wave) caused by the spread of the stimulus *towards* the electrode through the septum, followed by a large downward deflection (called the S wave) caused by the spread of the stimulus *away* from the electrode through the left ventricular mass. The result is an rS or right ventricular complex.

(An initial downward deflection is termed a Q wave; an initial upward deflection is termed an R wave; the ensuing waves are named by the succeeding alphabetical letters.)

NOMENCLATURE AND LOCATION OF UNIPOLAR ELECTRODES

All unipolar leads are termed V leads and are divided into extremity or limb leads and precordial or chest leads.

EXTREMITY OR LIMB LEADS

Extremity leads are normally of a low electrical potential and are therefore instrumentally augmented in electrocardiography. Such leads are termed augmented extremity leads and are prefixed by the letter 'A'.

AVL is the augmented unipolar left arm lead (Fig. 9A) and may be considered to 'face' the heart from the left shoulder (Fig. 9B).

AVF is the augmented unipolar left leg lead (Fig. 9A) and may be considered to 'face' the heart from the left hip (Fig. 9B).

PRECORDIAL OR CHEST LEADS

These leads (Fig. 10) are designated only by the letter 'V'.

V1 is located over the fourth intercostal space to the right of the sternal border.

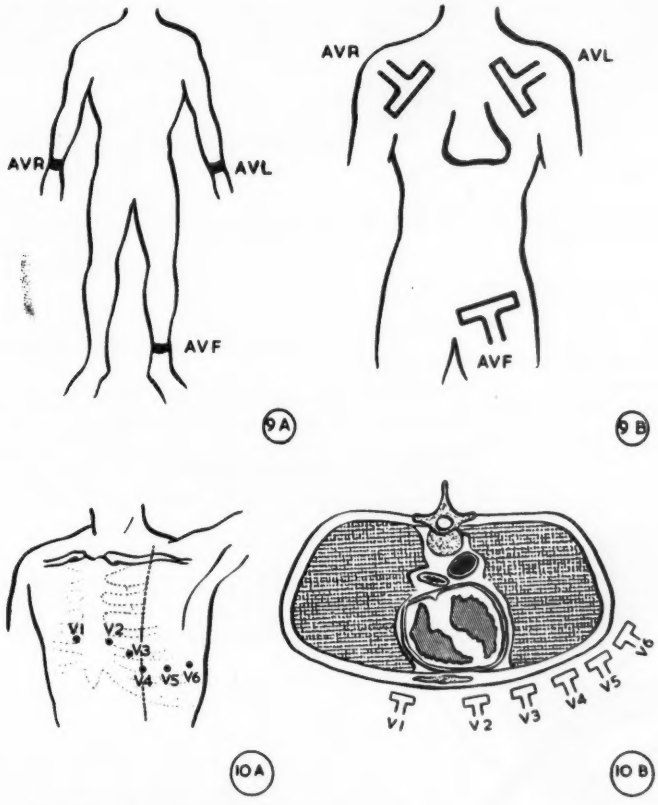


Fig. 10A. Precordial, Chest or V leads.
Fig. 10B. A transverse representation of the V leads in Fig. 10A.

AVR is the augmented unipolar right arm lead (Fig. 9A) and may be considered to 'face' the heart from the right shoulder (Fig. 9B).

V2 is located over the fourth intercostal space to the left of the sternal border.

V3 is located midway between V2 and V4.

V4 is located in the midclavicular line over

the interspace under which the apex of the heart is located.

V5 is located over the anterior axillary line on the same level as V4.

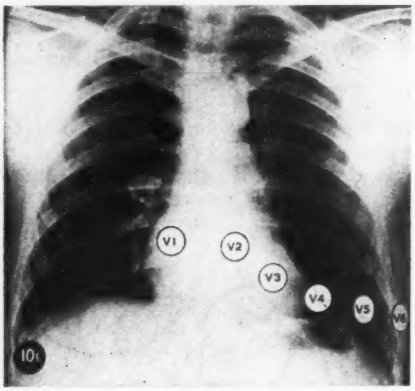


Fig. 10C. Precordial leads (*Cf.* Fig. 10A).

V6 is located over the midaxillary line on the same level as V4 and V5.

ELECTRICAL POSITION OF THE HEART

The anatomical position of the heart varies under normal or pathological conditions. These variations, however small, will be reflected electrically by the electrocardiogram.

Fig. 11. **Rotation round the antero-posterior axis.**

HORIZONTAL AND VERTICAL POSITIONS

Rotation round the antero-posterior axis, which runs through the centre of the septum of the heart from the anterior to the posterior surface (Fig. 11), will result in a vertical or a horizontal position.

HORIZONTAL POSITION

In this position the left ventricle faces the left shoulder and therefore lead AVL will show a left ventricular (qR) complex (Fig. 12). Conversely, lead AVF faces the right ventricle and will show an rS complex. *It is important, however, in determining the horizontal or vertical positions of the heart, to base the findings on the location of the qR complex.*

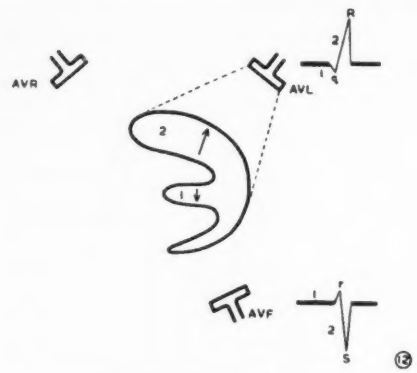


Fig. 12. Horizontal position of the heart.

VERTICAL POSITION

In this position the left ventricle faces the left hip and therefore lead AVF will show a left ventricular (qR) complex (Fig. 13).

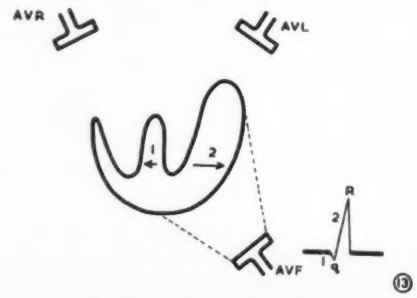


Fig. 13. Vertical position of the heart.

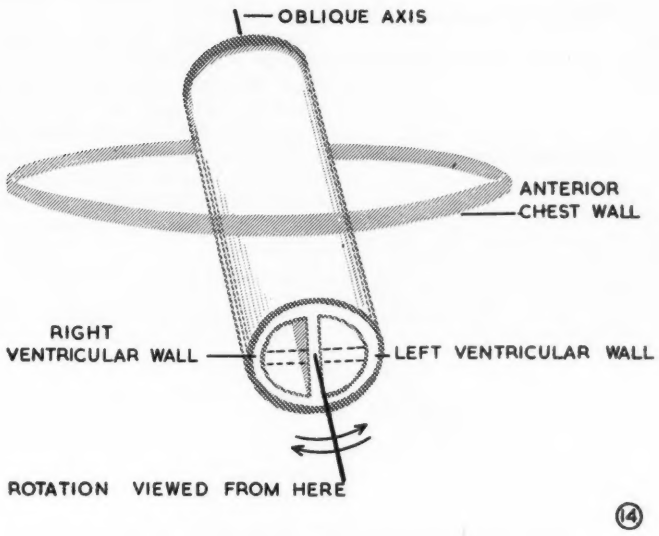


Fig. 14. Diagrammatic representation of the heart with the apex removed, to show rotation round the oblique axis.

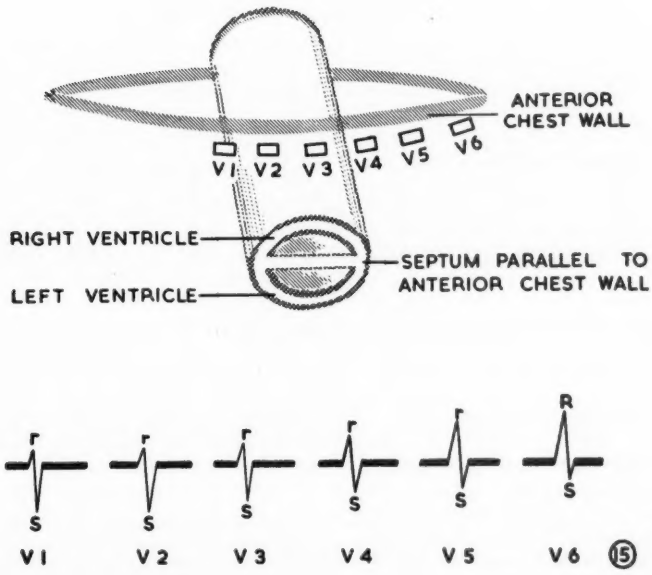


Fig. 15. Extreme clockwise rotation (diagrammatic).

To summarize:

A qR complex in lead AVL indicates a horizontal position.

A qR complex in lead AVF indicates a vertical position.

CLOCKWISE AND COUNTERCLOCKWISE ROTATION

The longitudinal axis of the heart runs obliquely from the apex to the base of the heart (Fig. 14). Rotation round this axis is conventionally viewed from below the heart looking towards the apex. This rotation is clockwise or counterclockwise.

COUNTERCLOCKWISE ROTATION

This will cause the left ventricle to rotate through a few degrees anteriorly so that now both the right and left ventricles face the anterior chest wall and the precordial leads (Fig. 16). The interventricular septum tends to lie at right angles to the anterior chest wall. Leads V1, V2 and V3 will face the right ventricle and record rS patterns. Leads V4, V5 and V6 will face the left ventricle and record qR patterns.

To summarize:

Extreme clockwise rotation: rS patterns in V1, V2, V3, V4, V5 and V6.

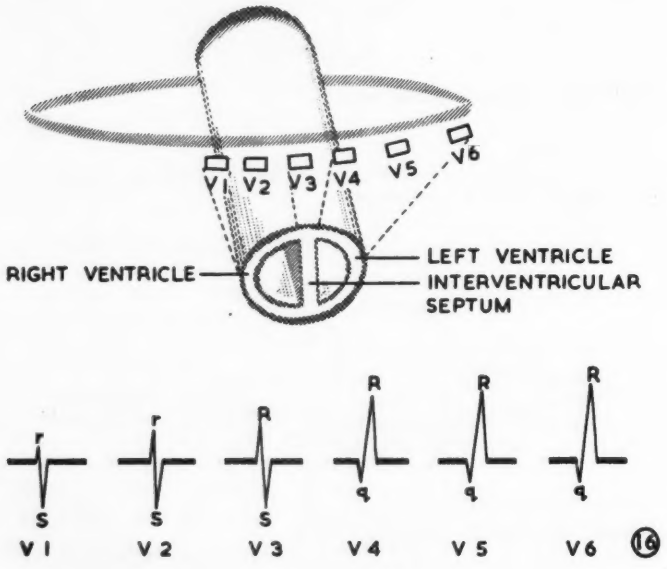


Fig. 16. Counterclockwise rotation (diagrammatic).

CLOCKWISE ROTATION

Clockwise rotation will cause the right ventricle to assume an anterior position. The right ventricular wall and the interventricular septum will lie parallel to the anterior chest wall. In extreme clockwise rotation (Fig. 15) the precordial leads V1 to V6 face the right ventricle and will record right ventricular or rS complexes.

Counterclockwise rotation: rS patterns in V1 V2 (V3). qR patterns in (V3) V4 V5 V6.

OPSOMMING

Die basiese beginsels wat ten grondslag van die hart se elektriese bedrywigheid lê, word uiteengesit.

Die manier waarop die patroon wat deur eenpoligeleidrade aangeteken word deur die anatomiese posisie van die hart bepaal word, word verduidelik.

(To be continued)

CALCIFIED CYST OF THE SPLEEN

T. SCHRIRE, F.R.C.S.
Cape Town

Calcified non-parasitic cysts of the spleen are a comparatively rare pathological finding. A review of the literature[3] revealed that, from 1827 to 1950, 183 cases of splenic cyst had been recorded and of these only 31 had become calcified.

Since 1950 several more of these rarities have come to light. Stahl and Stahl[7] reported a second case in their own practice, and Neidhardt,[4] while contributing still another, reviewed the literature once more and added the 2 cases of Tamaki[8] and of Lustok and Baum,[2] thus bringing the total to 35. The case here described is the 36th in the literature.

Shawan[6] described 4 forms of splenic calcification:

i. Small diffuse calcification, e.g. phleboliths.
ii. Large calcified tumours.
iii. Calcification in atheromatous splenic vessels.
iv. Calcified cysts.

Cysts of the spleen form a very interesting group. They are fully discussed by Fowler, who classifies them into true and false varieties. The true cysts differ from the false in that they possess a serous lining.

Pepere quoted by Fowler (*loc. cit.*) considers that 'serous cysts of the spleen originate from cellular nests which remain under the splenic capsule because of abnormal invagination of the peri-splenium during the development of the organ'. Anyone who has observed the lobulated spleen in some lower animals, the not infrequent subdivision of the human spleen with several splenunculi bunched together in the left upper quadrant or the more common occurrence of clefts in the anterior and posterior borders of the normal spleen, cannot fail to believe that the theory of Pepere deserves support.

This theory likewise explains the occurrence of such cysts at the margins of the spleen where fusion of the primitive lobes takes place latest. The lining of such cysts with epithelium similar to that of the peritoneum support Pepere's theory and the presence of such epithelium in our specimen leads one to the conclusion that this is a case of a splenic dermoid. Such dermoid cysts may be calcified or uncalcified and, like many inclusion dermoids elsewhere, they contain fluid, full of glistening cholesterol crystals.

The diagnosis of such a calcified mass presents many difficulties. The hydatid or echinococcal splenic cyst is said by Fowler to be 4 times as common as any other cyst and examples of these are to be found in most surgico-pathological museums. I have encountered a calcified hydatid of the spleen on the only occasion when I had the good fortune to operate on a splenic hydatid and personal questioning of my colleagues at Groote Schuur Hospital has indicated that only one other case of hydatid of the spleen, not calcified, has been found over the last 20 years.

Other calcified cysts or tumours must be considered, and renal cysts need particularly careful differentiation. Cysts of the mesentery may likewise become calcified on rare occasions, and a massive tuberculous gland or aggregate of glands also enters into the differential diagnosis.

CASE REPORT

Mrs. E. D., aged 43, was referred to me by Dr. H. Berelowitz. She complained of wind and of an aching pain in the left side of the abdomen for the last 18 years. At that time she was about to be married and on examination was found to have a lump in her left hypochondrium. This lump had not grown in size in the last 18 years, during which she had lived a normal life and had borne 2 children. She would not consent to have anything done about it. As the years passed, the lump appeared to come out from under the costal margin and to become more easily palpable. It was not tender and the patient, who had been born in Europe, had never been associated with dogs or with sheep. Her only complaint was that when constipated there was a sensation of pressure in the lump.

There had been no nausea, no vomiting and no history of tuberculosis. Her digestion and micturition were normal and her periods had always been regular.

She was a healthy, spare woman. Her blood-pressure was 125/80 mm. Hg., and her heart, chest and central nervous system were normal. A spherical mass about 10 cm. in diameter was easily palpable in the left hypochondrium. It moved freely on respiration and could be held

down easily. It could not be shifted across the midline over to the right side. The mass felt cystic, was slightly tender and could be ballotted in the loin. It was dull on percussion. When the patient stood up, the mass descended and could be felt below the level of the umbilicus just behind the anterior abdominal wall.

In 1940 and 1947 intravenous pyelograms had been done and showed a calcified spherical shadow (Fig. 1) which was unrelated to the left kidney and lay anterior to the bodies of the vertebrae. A renal origin was therefore excluded and a barium meal investigation was done in 1954. This showed that while the mass lay close to the ascending and descending loops of the splenic flexure, it was not attached to the colon and showed no signs of originating from this or from any other part of the gastro-intestinal tract. The chest was normal.

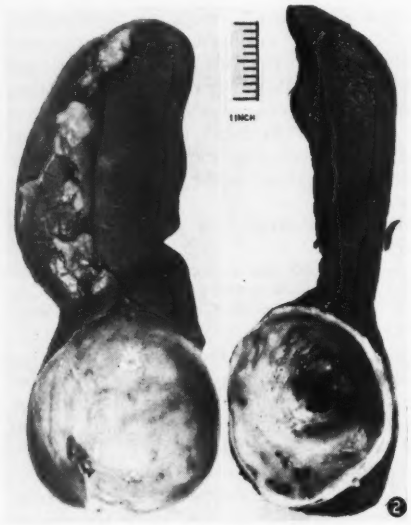
A blood examination revealed:

Erythrocytes: 4,680,000 per c.mm.
Haemoglobin: 13.9 g. %.
Leucocytes: 5,200 per c.mm.
Polymorphs: 72%.
Lymphocytes: 22%.
Large mononuclears: 3%.
Eosinophills: 3%.

The platelets appeared normal in shape and number and no abnormal or immature leucocytes were seen.

DISCUSSION

The absence of any history of association with dogs or sheep was against a diagnosis of hydatid disease. In our hands the Casoni test has never been very reliable and because calcified hydatid cysts are usually regarded as dead hydatids, not evoking antibody reactions, further tests were not pursued. However, the lungs were X-rayed and found to show no



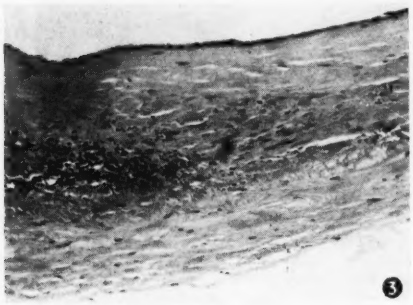
evidence of echinococcal or of tuberculous disease, and a careful examination of the rest of the body disclosed no other cysts.

The alternative diagnosis of a calcified mesenteric cyst was not a very firm one. Cysts of the mesentery, unless tuberculous, do not as a rule undergo calcification, and a tuberculous cyst of such a size implied that a large mass of caseous mesenteric glands had broken down with subsequent calcification. There was no other evidence of tuberculosis to be found and it was difficult to imagine that a suppurating mass of tuberculous glands of such a size should exist as a solitary manifestation of the disease.

The possibility of an ordinary calcified mesenteric cyst could not be excluded entirely and it was necessary to consider where such a cyst might arise. The mesentery is well known as a common site for such cysts, but its situation in the left hypochondrium was a little too high for this structure. The splenic flexure, however, was seriously considered as

a possible site of origin and one of our skiagrams demonstrated the cyst to be lying squarely on this flexure and gave support to this opinion. In view of the possibility that the cyst might originate in the mesentery of the colon and that its excision might force us to do a colectomy, the patient was put on to a full pre-operative colonic preparation.

Operation was performed on 12 May 1955. The abdomen was opened through a left paramedian incision and a cystic mass about the size of a closed fist was found occupying the



lower pole of the spleen (Fig. 2). The spleen itself was elongated, evidently by the constant drag of the mass. No other cysts were present in the abdomen, and the liver, gall bladder and duodenum were normal. The spleen was mobilized by incising the peritoneum on its lateral side and stripping it inwards by entering the pre-aortic space. The short gastric vessels were tied and cut, the pedicle of the spleen was subdivided, the vessels were tied and cut individually and the spleen was removed. The operative field was dry and the abdomen was closed.

Convalescence was uneventful, the patient getting up on the second day and being discharged one week later.

Dr. G. Selzer reported as follows:

'The specimen is an elongated firm spleen weighing 240 g. At the lower pole is a well-circumscribed cyst 6 cm. in diameter. The content is a glistening greyish-yellow fluid. The glistening appearance is due to a vast accumulation of cholesterol crystals. Several areas of calcification are present in the wall.

Histologically (Fig. 3) no hooklets or scolices are demonstrable, nor is the appearance of the wall that of a hydatid cyst. The wall consists of dense collagenous tissue and the inner lining is a layer of flattened cells.'

SUMMARY

1. A case of a calcified non-parasitic cyst of the spleen (the 36th in the literature) is recorded.

2. The literature on calcification of the spleen and on cysts of the spleen is reviewed.

OPSOMMING

1. 'n Geval van 'n verkalkte nie-parasitiese geswel van die milt (die 36ste in die literatuur) word aangeteken.

2. Die literatuur insake verkalking van die milt en geswelle van die milt word hersien.

I would like to record my thanks to Dr. H. Berelowitz for referring the patient to me and for giving the anaesthetic, to Dr. M. Pimstone for assisting at the operation, and to Dr. G. Selzer of the Pathology Department of the University of Cape Town for the pathological report. I would also like to thank Mr. G. McManus of the Department of Surgery who kindly prepared the illustrations.

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SKELETAL CHANGES IN ENDOCRINE AND METABOLIC DISORDERS

VII. HYPOPARATHYROIDISM

W. P. U. JACKSON, M.D.

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Hypoparathyroidism is most commonly produced by removal of the parathyroid glands during thyroidectomy. Damage to these glands is often temporary and produces only transient tetany, but their removal causes permanent hypoparathyroidism.

'Idiopathic hypoparathyroidism' is a rare serious disorder in which all 4 glands atrophy for no known reason. In this way it resembles that variety of Addison's disease due to idiopathic adrenal atrophy and, in fact, the two diseases are occasionally combined in one patient.

Tetany is the outstanding symptom, some-

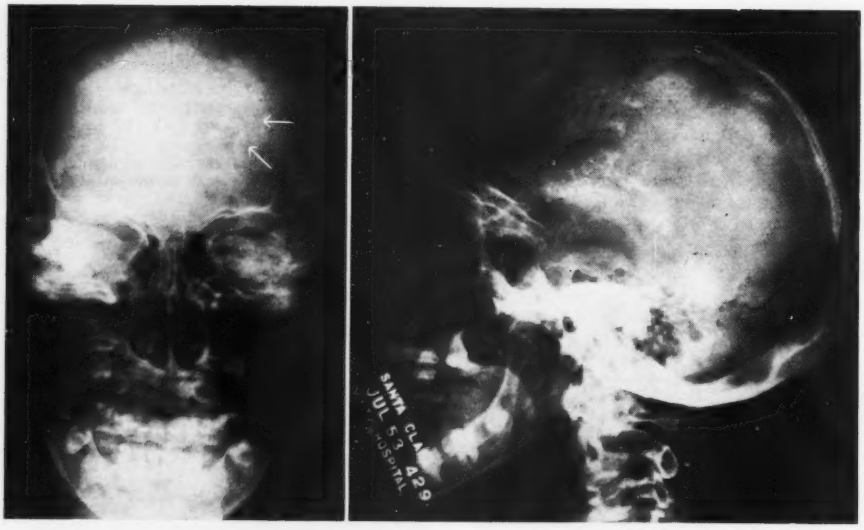

Fig. 1. Skull of a girl aged 23, showing calcification of basal ganglia and underdeveloped tooth roots.

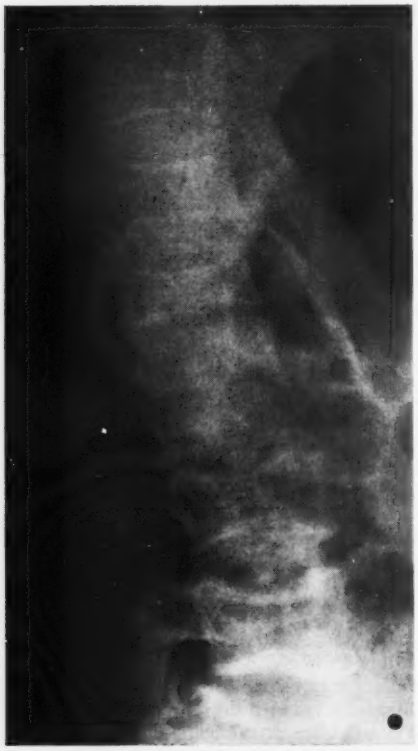

Fig. 2. Decalcification of the spine, as in this case, with multiple vertebral collapse, is a rarity.

times with epileptiform convulsions. The serum calcium is low (when below about 7.5 mg. per 100 ml., calcium disappears from the urine), the serum phosphorus is raised and the alkaline phosphatase is normal. The reabsorption of phosphate by the renal tubules is high. Other phenomena seen in long standing cases include cataract, multiple ectodermal atrophies, secondary moniliasis of the nails and papilloedema.

Changes to be found on X-ray are usually limited to the head (Fig. 1). Bilaterally symmetrical, punctate calcification of the basal ganglia (and sometimes of the cerebellum) may be found. If the disease occurs in childhood the permanent tooth roots remain underdeveloped. The skeleton may have an increased density, though rarely decalcification is discovered (Fig. 2). This has been considered on theoretical grounds to be due to osteomalacia. Bone biopsy in one case, however, did not bear this out.

OPSOMMING

Hipoparatiroïdisme kan primêr (atrofies) of sekondêr —volgende op snykundige verwydering—wees. Tetanie is die gewone waarneembare simptoom.

Daar is min X-straal-veranderings. Die opvallendste is punktaat-verkalking van die harsingboomsenuknoop.

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INGUINAL HERNIA

I. THE DEVELOPMENT OF THE SURGICAL TREATMENT†

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Pretoria

As a prelude to Part II of these reports, it is of interest to give a brief review of the development of the surgical treatment of inguinal hernia. It is virtually impossible to cover the voluminous literature on the subject and a limited number of seemingly representative and informative articles have been used in this compilation. Two works, one by Zimmerman and Anson[1] and the other by Edwards,[2] have proved particularly valuable. The illustrations have been redrawn from articles referred to in the text, sometimes with modifications.

THE PRE-ANTISEPTIC PERIOD†

By 900 B.C. the Phoenicians treated inguinal hernia by girdle and compresses. By 400 B.C. Hippocrates had described inguinal hernia. The term 'hernia' is derived from the Greek *hernios*, a branch or offshoot. In the first century A.D. Celsus practised ligature excision of the unopened hernial sac, but advised against operating for strangulated hernia. In the second century Heliodorus carried out ligature and excision of the freed sac, and in the fourth Oribasius applied the cautery after such operation to promote the ultimate formation of fibrous tissue. In the seventh century, Paulus Aeginata ligated and excised the freed sac.

Little if any advance occurred in the treatment of inguinal hernia during the Dark Ages. Strolling herniotomists extracted a toll of testicles, often fed to trained dogs to support the claim of operation without castration, while bloody dressings and hasty departures—sometimes with relatives in hot pursuit—were usual. Among these charlatans were others, like the Norcinis of Central Italy, who kept the methods of herniotomy handed down from father to son as a trade secret. It is said that Frère Jacques operated upon over 2,000 hernias with attendant orchidectomy. By the 16th century Paré was employing a method devised by Geraldus of Metz, slipping a ligature round the neck of the sac and tying it over a bone plate with the object of causing it to slough and subsequently obliterate. The use of various forms of truss, some filled with quaint medicaments, remained the main form of treatment.

Pierre Franco, a contemporary of Paré, advised operation for relief of strangulation, formerly treated by taxis, clysters, bleeding, purging, compresses and ointments. He incised the constricting ring. Strangulation became for many surgeons the only indication for operation. At the time of Pott various operations carried a mortality of over 30%. de Garengot devised a procedure for dissecting out the sac, rolling it into a pad and suturing it in the canal.

The unsatisfactory state of affairs lasted well into the 19th century, except that by this time the results of the valuable anatomic studies of Vesalius, Scarpa, Cooper, Gimbernat and Hesselbach were available. Attempts were made to promote cure by inducing sepsis, seton treatment (Wutzer, 1840), injection of iodine (Pancoastin, 1844) or application of iodine to the opened sac. In 1857 Wood reintroduced subcutaneous operation for ligature of the sac, but the modern surgery of inguinal hernia required the introduction of antiseptic and aseptic technique before it could advance.

HIGH LIGATION OF THE SAC AND VARIOUS METHODS OF REPAIR

In 1869 Marcy of Boston, the first American pupil of Lister, emphasized the importance of high ligation of the sac and performed for the first time suture of the transversalis fascia and closure of the internal inguinal ring. He devoted himself largely to the problems of hernia. By 1881 Marcy employed kangaroo tendon to effect the repair. In 1886 MacEwen re-employed the operation of de Garengot, who had used the rolled-up sac as a tampon to be stitched into the internal ring, but brought the sutures holding the rolled-up sac through the muscles and skin above the internal ring and then tied them. By 1889 Edoardo Bassini, Professor of Surgery at Padua, prob-

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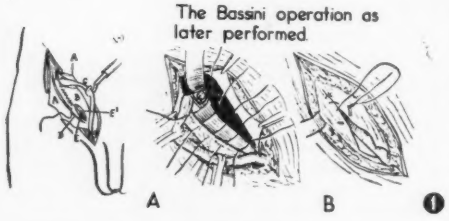
† References will be listed at the end of the second communication.

ably stimulated by hearing Marcy at an International Medical Congress in London in 1881, and Halsted had independently developed their operations. The essential features of these are shown in Figs. 1, 2. It will be noted that both operations sought to reinforce



Synopsis of the Development of the Modern Surgery of Inguinal Hernia

Marcy (1869): Suture of transversalis fascia and closure of internal inguinal ring.

Fig. 1. Bassini (1889): Suture of transversalis fascia, transversus abdominis and internal oblique muscles to inguinal ligament (Figs. 1A, 1B.)
A = External oblique; B = Internal oblique; E, E1 = Transversalis fascia and transversus muscle; D = Inguinal ligament.

Fig. 2. Halsted (1889): Meticulous anterior to the cord repair.

It is instructive to note that the operation actually used by Bassini effected a more meticulous closure than that usually taught as the 'Bassini' operation. After division of the fascial floor of the inguinal canal from the internal ring, the transversalis fascia and transversus abdominis (Fig. 1, E-E[1]) and the internal oblique (B) were carefully sutured to the shelving portion of the inguinal ligament D.[1] 'C' is the spermatic cord and 'A' the cut external oblique, which was resutured anterior to the cord.

In 1895 Andrews modified the Halsted procedure (Fig. 3). There were several other variations based on the Bassini-Halsted principles. Fowler (1897) used intraperitoneal transplantation of the cord. Ferguson (1899) sutured internal oblique and conjoined tendon anterior to the cord. Kocher (Fig. 4) transposed the sac obliquely into the abdominal wall.

From these endeavours it became possible, at the opening of the present century, for the average operator to deal satisfactorily with most inguinal hernias. Little more than a decade earlier these 'had baffled the efforts of the best surgeons to cure it' (inguinal hernia).[3] In 1905 Scott used a relaxation incision through the anterior rectus sheath to release tension on the suture lines of the inguinal reinforcements. In 1906 Torek added dissection of the cord and separation of the vas deferens from the vessels. There is no doubt that this manoeuvre made it possible for the first time to visualize clearly the actual neck of the sac of an oblique inguinal hernia.

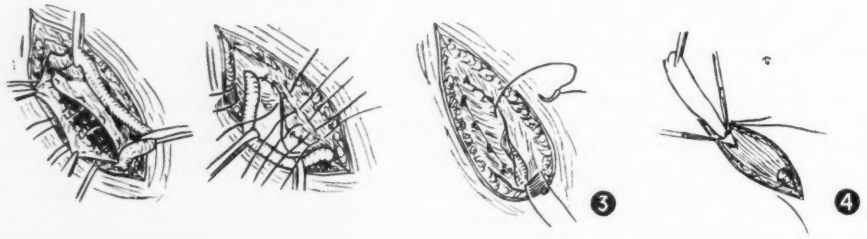


Fig. 3. Andrews (1895): Posterior and anterior to cord repa r.
Fig. 4. Kocher: Transposition of the sac.

the inguinal canal. In the Bassini operation the barrier to forward passage of abdominal contents was placed deep to the spermatic cord, in the Halsted superficial to it. In time Bassini followed up all but 4 of 251 cases, among whom there had been *no deaths*, for periods of from 1 month to 4½ years. He found 7 recurrences!

METHODS OF ADDITIONAL REINFORCEMENT OF THE INGUINAL REGION

It is obvious that some surgeons were not content with the above methods, no doubt because of an early appreciation of a recurrence rate after surgical treatment of inguinal hernia, or sometimes because of an individual

approach to the whole problem. In 1901 MacArthur[4] reported on the use of autoplastic suture in hernia and other diseases. He used strips of fascia derived from the external oblique aponeurosis as suture material in the repair of hernias. Others have since reported on similar methods.[5, 6] In 1898 Bloodgood, then Halsted's assistant, introduced suture of the rectus muscle to Poupart's ligament (Fig. 5) as reported by Halsted.[3] In 1911 Downes re-introduced the method. In 1909 McGavin used silver filigree inlay to construct a barrier to protrusion into the inguinal canal. In the following year Kirschner introduced the use in oblique hernias and included this between fascial sutures.

The method was most widely adopted by others to deal with post-operative recurrences of hernia. Its use also extended surgical treatment to inguinal hernias in patients over 45 who were usually treated with a truss into the early 1930's.[8]

Shortcomings of the procedure became apparent. Extremely capable surgeons reported a recurrence rate as high as 7.8%.[9] There had been many objections to the obvious damage inflicted by the very gross needle which carries the fascial sutures; the actual 'living' of the

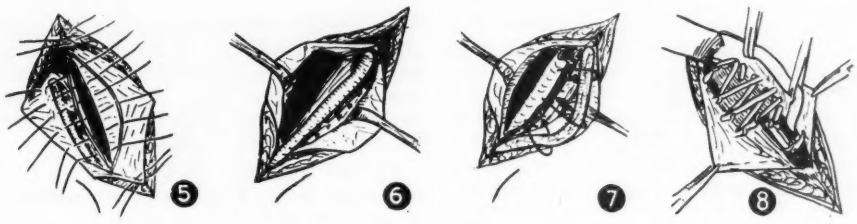


Fig. 5. Bloodgood (1898): Suture of rectus muscle to Poupart's ligament.
Downes reintroduced Bloodgood's method in 1911.
MacArthur 1901 used fascia from external oblique as suture material.

Fig. 6. Scott (1905): Relaxation incision through anterior rectus sheath to release tension on suture line of the modified Bassini repair.

Fig. 7. Berger's procedure.
Torek (1906): Dissection of the cord and separation of vas deferens and vessels.
Edmunds (1908): Abdominal approach for bilateral inguinal hernias.
McGavin (1909): Silver filigree inlay.

Fig. 8. Kirschner (1910): Closure of defect by transplants of fascia lata from the thigh.
Gallie (1921) popularized the method and laid the ghost of anterior to the cord repairs.
Hass (1931): Heterogenous transplants of ox fascia (in dogs); Chandy (1945) in humans.
Veal (1942): Osteoperiosteal graft from tibia.

of transplants of fascia lata from the thigh (Fig. 8), a method which slowly gained considerable favour after Gallie's report of 1921.[7]

Gallie claimed that the free fascial grafts lived and became parts of the body. He showed that the conjoint tendon and internal oblique which had been approximated to Poupart's ligament, separated it from the effects of tension. He advised that the fascial strips be placed under no greater tension than necessary to make them lie flat—they were to serve as a gap-filling filigree of living tissue and provide a sturdier barrier than the fibrous-tissue-filled metal filigree. He laid the ghost of the superficial-to-the-cord repairs and emphasized that it was vastly more important to stop a potential hernia from entering the inguinal canal than from leaving it. He also recognized the internal ring as the weak point fascia came under suspicion and the scar and muscle hernia in the thigh produced a new disability.

On the subject of 'living tissue' grafts it is of interest to add that the use of osteoperiosteal transplant in the treatment of inguinal hernia has been reported.[10] Heterologous grafts of ox fascia were studied in dogs by Hass in 1931.[11] A significant series of human inguinal hernias treated with specially prepared ox fascia has been reported.[12]

The use of silk for hernia repair was introduced by Sampson Handley in 1918. Ogilvie[13] advocated a strong lattice replacement of the inguinal mechanism with stout Chinese twist silk, again, like Gallie, to be placed 'firm but not tight'. Maingot[14] introduced floss silk—individual fibrils of natural silk—for the same purpose.

CLOSURE OF THE NECK OF THE SAC AND ITS IMMEDIATE SURROUNDINGS ONLY

In 1908 Edmunds used an abdominal approach for bilateral inguinal hernias. In 1913 Hull[15] advised a simple ligation of the neck of the hernial sac. In the same year Bates reported that he made a small incision, opened the aponeurosis of the external oblique, exposed the internal inguinal ring, defined the sac and opened the peritoneum at its neck. The internal oblique and transversalis fascia were pulled into the wound, closed by purse-string suture and the peritoneum repaired. In 1919 la Roque performed transperitoneal ligation of the hernial sac.

Some of these operations stressed the truly high ligation of the hernia and concentrated on dealing with the peritoneal defect. Some of them also, inadvertently or by design, narrowed the internal inguinal ring. A number of surgeons has continued to favour these methods, with a few technical modifications.[16-21]

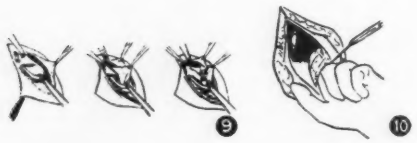


Fig. 9. Hull (1913): Simple ligation of the neck of the sac.
Bates (1913): Exposure of internal ring through small incision. He opened peritoneum at neck of sac and sutured it.
Sutton's (1936) procedure is illustrated.

Fig. 10. Collens' (1942) procedure.
Sampson Handley (1918): Silk repair for hernia.
Ogilvie (1937): Strong lattice of Chinese twist silk.
Maingot (1941): Individual fibrils of natural silk.

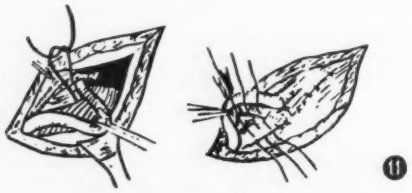


Fig. 11. MacGregor (1930): Repair of internal ring, reinforcement by slip of aponeurosis and some displacement of the cord.

RECURRENCE OF HERNIA AFTER OPERATION

The Lessons of the Second World War

It has already been mentioned that the multiplicity of methods used in the operative treatment largely arose because of greater or lesser hernia recurrence rates. Reports of some statistical value appeared in the literature from the 1920's onward.[2, 22-30]

In 1937 Ogilvie[13, 31] recognized 3 types of inguinal hernia requiring different methods of operative treatment:

1. Presence of a sac only—removal of sac alone;
2. Above, plus stretching of the internal ring —removal of sac and plastic repair of the ring;
3. Large oblique and direct inguinal hernias with broken-down sphincter mechanism—strong lattice repair of the inguinal region.

The recruiting and drafting of men for the armed forces brought to light the urgency of the problem of inguinal hernia. Figures of incidence as high as 11% in batches of recruits were recorded. Recurrence after operation disabled many men in training or combat. In one year (1942) 805 operations were carried out for recurrences in military hospitals alone. Nearly all of these had been operated on for the first time before enlistment, mostly for hernia of the indirect type. In one series of 131 recurrences after operation for indirect hernia the following types of recurrence were noted:[2]

Fig. 12A—D. Lytle (1945): Detailed study of the internal ring. Repair of the internal ring.

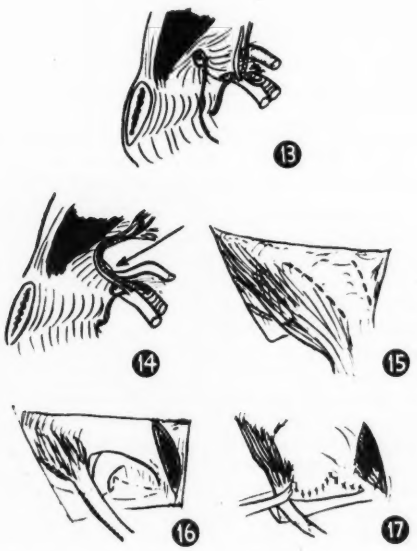
1. *Indirect Hernia*: A new sac passing through the internal ring—98 cases;

2. *Direct Hernia*:

(*a*) Diffuse bulge with a saucer-shaped sac—20 cases;

(*b*) Funicular sac through an opening in the transversalis fascia just lateral to the insertion of the conjoined tendon—31 cases.

It was felt that fewer recurrences developed after the use of non-absorbable sutures. However, there was a high incidence of delayed sepsis in cases in which these were used in British Military and Emergency Medical Services and in overseas hospitals. Such was the irony of fate that Ogilvie, the Consulting Surgeon to the British Forces in the Middle East, had to ban the use of silk under service conditions. Many of us could not suppress sardonic humour at receiving these instructions, after having been taught by him to use twisted silk and subsequently having been forced to assist the natural removal by suppuration of feet of the material.



Figs. 13—17. McVay and Anson (1949): Detailed study of indirect hernia (arrow in Fig. 13), and direct hernia (arrow in Fig. 14). Relaxing incisions are indicated in Figs. 15—17 and suture of aponeurosis to Cooper's ligament and fascia in Fig. 17.

It also emerged that the sooner operation was carried out after the hernia appeared, the better the chances of cure. The older the patient at the time of operation, the greater was the chance of recurrence. Recurrence was less frequent in men with good musculature.

Closure of the sac alone, leg-raising exercises in bed from the first post-operative day and relatively early ambulation became the practice in most cases in British and Dominion service hospitals.

In the meantime, information obtained by painstaking dissections during the preceding years was gaining wide-spread publicity. The mechanisms of the inguinal region had been extensively studied.[32-34]

The application of the Lotheissen operation as performed for femoral hernia (and advocated for inguinal hernia by Babcock in 1927[35]) came into vogue with modifications in the 1940's.[36, 37] The present standardized form of this operation is generally referred to as the McVay operation (Fig. 13). Various methods of strengthening the defect in the internal oblique, transversus muscle and transversalis fascia were also gaining favour and the essential differences between the operative problems of direct and indirect inguinal hernia, stressed by several authors,[38-40] were becoming common knowledge.

THE POST-WAR PERIOD

Despite continued work on the mechanisms of the inguinal region, the experiences of the war did not lead the surgery of inguinal hernia as far as had been anticipated. Recurrence remains a problem[41-47] and there are, no doubt, many factors responsible for this. The further reports on anatomical studies[48, 49] have not added greatly to the results of operation. The results depend more on the skilled use of any particular operation than on the type of operation used and although, on the whole, direct and indirect inguinal hernias are considered to require different types of operation, a procedure theoretically adequate for indirect hernia may be followed by the appearance of a direct hernia as a 'recurrence' or, more rarely, by femoral hernia. Also the presence of a diffuse bulge in the inguinal region after an operation for hernia is just as disappointing to the patient as is a formally classified hernia. This last has been noted commonly after the methods which have stressed the non-tightness of repair.[8]

It would now seem at first sight that the surgeon is faced with an endless choice of

operations, some very simple, some elaborate. They range from simple exposure of the sac and its high ligation as first advocated by Hull (and most eminently suited for infants, children and perhaps some young adults) through various forms of narrowing of the stretched internal ring (the most common usage in the British schools and their offshoots is probably that associated with the name of Lytle[20]) to various procedures which, as it were, build a barrier to prevent the entrance of a protrusion into the inguinal region. To effect this purpose inlays of skin taken from the abdominal incision and deprived of subcutaneous tissue, after the manner usually ascribed to Mair[50] but also reported on by others (*inter alios*[51, 52, 53]) are favoured by some, despite reports of a percentage of ill effects from burying of cutaneous tissue.[54] Others resort, at least on occasions, to the use of fascial sheet inlays,[55] or metal filigree in which tantalum has replaced the silver filigree used by McGavin in 1909. Fascial sutures are still used but have been discarded by most operators. Some stress the nature of suture material to build the barrier—silk, floss silk, nylon, stainless steel wire, tantalum wire—a few use material from the sac.[56] In some hands, especially for recurrent hernias in old men, removal of testis and cord has found favour, while others divide the cord as an aid to operations on selected types of inguinal hernia, as described at least 20 years ago.[57]

The fact, however, remains that most surgeons for most cases continue to rely for the 'barrier' on the transposed musculo-aponeurotic layers of the abdominal wall. Some have been converted to the McVay procedure, but the majority still, in effect, perform some modification of the Bassini operation, most usually with a medial tension-relieving incision, which manoeuvre they refer to as a 'Tanner slide', after the distinguished London surgeon.

THE INJECTION TREATMENT

For a brief spell during the later 1930's and early 1940's there was a passing vogue in some quarters for the substitution of surgery by the introduction of sclerosing solutions into the sac of hernias after reduction of the contents. It soon became evident that large numbers of injections were required to gain results, the recurrence rate was very high and serious complications were not uncommon.[58-83] The method died a not untimely death.

A SERIES OF OPERATIONS

Surgical operation remains the only effective treatment for inguinal hernia. In current local practice the Bassini type operation, frequently modified in some way (most usually by the 'Tanner slide') remains the most commonly used method. This is shown in Table I, which analyses the types and number of operations effected in the professorial surgical unit of the Groote Schuur Hospital over the 4-year period 1950-53.

TABLE I: 536 CONSECUTIVE OPERATIONS FOR INGUINAL HERNIA

Bassini-type (usually with Scott slide)	371	(70%)
Herniotomy alone	87	(16%)
Lytle	26	(14%)
Mair	21	
McVay	20	
Hernioplasty by grafts or tantalum mesh	10	
Kocher	1	
Total	536	

In interpreting the terminology used: *herniotomy* indicates high ligation of the sac only; *Bassini*, etc. is by now obvious; *Lytle* means the procedure indicated in Fig. 12D; McVay that indicated in Figs. 15-17. The Lytle operation is based on the concept of the internal ring in Fig. 12A acting as a shutter mechanism as in Fig. 12B, becoming stretched by descent of an *indirect hernia* as shown by the arrow in Fig. 12C which crosses the inferior epigastric artery. The operation is applicable only to indirect hernia before the musculofascial inguinal floor has become weakened and attenuated. The McVay operation is applicable to either an *indirect hernia* which protrudes into the abdominal wall as shown in Fig. 13, or a *direct hernia* which bulges medial to the epigastric vessels, as shown in Fig. 14.

OPSOMMING

Die geskiedenis en ontwikkeling van die behandeling van liesbreuk is in die eerste deel beskryf. Die werklike baanbreker in die moderne chirurgie van hierdie kwaal was Marcy in die Verenigde State van Amerika. Sy werk is toe opgeneem en verder ontwikkel deur Bassini van Italië en van dié tyd is daar menigvuldige operasies ontwikkel.

In die tweede deel is 'n series van gevalle beskryf saam met opvolging daarop.

THE EFFECTS OF SUBTOTAL GASTRECTOMY

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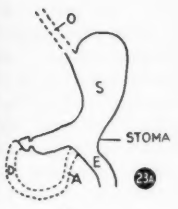
Department of Surgery, University of Cape Town

(*Concluded from p.* 39)

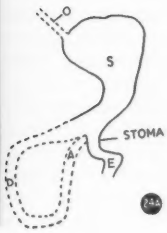
I. DUMPING SYNDROME

It is often stated that these symptoms are due to distension of the gastric stump,[39, 49, 54] but experimental distension of the stomach cannot reproduce them and it is generally accepted that they arise from the hyperperistalsis induced by the rapid entry of a bulky meal into the efferent loop.[14, 43, 77, 98, 99, 107] The dumping syndrome may consequently occur in normal people with rapid gastric emptying,[4] and a gastro-jejunostomy, by producing a dependent stoma (Fig. 23), will result in very rapid emptying (Fig. 24), which often produces symptoms.[55, 70, 78, 85, 86] This rapid passage of food eaten can be confirmed by fluoroscopy[85] and the symptoms can be provoked by any mechanical, physical or chemical stimulation of the jejunum[4] such as the introduction of iced water[19, 24] and hypertonic fluids[4, 23, 48] into a jejunostomy tube. It follows that the symptoms only manifest themselves once the patient resumes a normal diet[48] and even then they may only occur after the biggest meal of the day or perhaps only after an exceptionally large meal.[16, 43, 48] It has already been pointed out that the higher the gastric resection, the more precipitate the gastric emptying, so it is not surprising that dumping symptoms are more common and more severe the higher the gastrectomy.[32, 36, 43, 70, 102]



It is not only the bulk of the food eaten which is responsible for this effect, but if the food is hypertonic it will attract fluid from the bowel wall into the lumen and so further increase the bulk in the bowel.[48, 102] It is thus understandable that a meal with a high carbohydrate content will be more likely to produce symptoms.

It has been claimed that dumping symptoms are due to the drag on the oesophagus by filling of the unsupported gastric stump and that the introduction of a weighted balloon into the stomach will reproduce the symptoms.[16] During my attempts to confirm this, I found repeatedly that the weight immediately slipped into the efferent loop, demonstrating again the dependent drainage of the stomach. The presence of the balloon in the efferent loop may form a stimulus for hyperperistalsis and the production of symptoms,[85] which may be an explanation for the original reports.



Rapid filling of the jejunum can be confirmed by finding distension of the bowel[32] which is sometimes encountered and another confirmatory test is a glucose tolerance curve

which will show hyperglycaemia due to the extremely rapid emptying and absorption of the carbohydrate.[48, 85]

This condition will tend to make the patient eat smaller meals and may thus be an important factor in the common post-operative loss of weight.[2, 63, 86, 102] Its chief significance remains the symptoms which the patient experiences, however, and it is important to keep in mind that if the operation is performed for minimal complaints, the patient will justifiably be dissatisfied if he exchanges a virtually asymptomatic state for these post-operative symptoms. This is sometimes inevitable if the gastrectomy is performed for haemorrhage (occurring before very significant symptoms), but in elective operations it is wise to assess the patient very carefully before deciding on gastrectomy, to ensure that the patient 'has earned his gastrectomy'.

The incidence of the dumping syndrome varies in different reports depending on the strictness of the criteria adopted for making the diagnosis[36] but is generally in the region of about 10%.[16, 22, 43, 53, 75, 90, 92, 95, 102] It is less common[16, 20, 43, 65, 69, 102] and less persistent[92] after a Billroth I operation but it has already been shown that a high Billroth I resection may also produce dumping.[32, 82] The use of a valve in the Polya type of operation has no effect on the incidence of this complication.[16, 32]

To avoid confusion it is important to differentiate between mild and severe dumping syndromes, as they differ in their significance, treatment and prognosis.

i. Mild dumping symptoms are very common and probably most patients experience them to a greater or less degree some time after the operation. In this series 13 patients have been seen with minor dumping syndromes following gastrectomy and 3 following posterior gastro-jejunostomy.

These symptoms may be no more than an unpleasant awareness of intestinal activity[43] (4 cases) or, if the hyperperistalsis takes the form of excessive segmental movements only, the resultant increased absorption and decreased propulsion will produce constipation (1 case) often improved by atropine. On other occasions the propulsive waves are accentuated, resulting in very rapid passage of food along the bowel, reaching the caecum in 5 minutes[100, 102] and a diarrhoeic stool 15 minutes after the meal[48] (1 case). The commonest complaint, however, is abdominal cramps shortly after the meal (9 cases).

These mild dumping symptoms usually improve with time and are readily treated by the advice already mentioned to ensure slow gastric emptying,[32] viz. frequent, small, solid, meals with a period of recumbency after the meal; the food should not be iced[4] or hypertonic. This simple advice, plus reassurance, is sufficient to relieve almost all mild cases and only exceptionally is it necessary to prescribe ganglionic blocking agents to reduce intestinal motility.

ii. Severe dumping symptoms are much rarer (only 6 cases have been seen personally) but much more important as they produce significant disability. For convenience this syndrome can be said to produce 3 types of symptoms; all may not always be present and if they are their severity may vary considerably. These symptoms are:

(*a*) Visceral sensations of increased peristalsis, i.e. colic, audible borborygmi, a feeling of fullness or even pain in the epigastrium,[48] nausea[48] and a diarrhoeic stool, all manifesting soon after the meal,[48, 85, 102] especially with a meal containing a high sugar content.[48] These symptoms are usually severe and persistent and almost invariably require a ganglionic blocking agent for adequate relief.

(*b*) Vasomotor disturbances, e.g. a feeling of warmth, sweating, palpitations, pallor, tachycardia, raised blood pressure and absence of the normal post-cibal rise of skin temperature.[9, 84, 85]

These symptoms and signs closely simulate the effects of adrenaline secretion[85] but they are not improved by adrenergic drugs:[85] on the other hand they are improved by ganglionic blocking agents,[102] splanchnic block[14] and thoraco-lumbar sympathectomy,[74] which suggests that this is a reflex via the sympathetic nervous system from the hyper-active bowel.

(*c*) In some cases a notable feature is extreme fatigue, muscle tiredness and weakness which may be so severe as to force the patient to lie down. These symptoms come on about ½ hour after the others and last for 1½ hours.[84] These unusual symptoms are often associated with electromyographic and electrocardiographic evidence of potassium deficiency[71, 84] although the alteration of the electrocardiogram also occurs with mere distension of stomach or small bowel[13, 41] particularly if the food is cold.[13, 103] In addition, a lowering of the serum potassium by about 1 m. eq. per litre during these symptoms has been reported,[85] although this has not always been confirmed.[71] It thus seems very probable that potassium depletion is the cause of this symptom, but the mechanism of this depletion is

rather uncertain. It may be that adrenaline secretion associated with the vasomotor disorder mentioned above is responsible[17, 38, 84, 85] but more probably it is due to intra-cellular binding of potassium with the extremely rapid deposition of glycogen following the excessive absorption of carbohydrate entering the bowel so precipitately.[34, 45, 84] It is thus a feature of this condition that it is associated with, but not caused by, a rise of the blood sugar.[48, 84] Confirmation of this theory of potassium depletion is the fact that intravenous potassium relieves the weakness[85] and the symptom can be prevented by taking a large amount of potassium before the meal.

As these dumping syndromes are due to rapid emptying of the gastric stump[48] they can be prevented by avoiding this complication of gastrectomy. A Billroth I operation (if not done too high) has been shown to be reasonably effective and this is thus yet another reason why this operation should be performed whenever possible. In addition, the gastrectomy must be done no higher than is necessary. It has been suggested that a small stoma will delay gastric emptying[25, 64, 83] but it probably has no effect.[32, 68] It has already been mentioned that low gastrectomy plus vagotomy and gastro-duodenal anastomosis[100] may possibly be the answer to this problem.

In the management of the mild dumping syndrome one should keep in mind that it improves with time and that simple measures to delay gastric emptying and prevent jejunal stimulation are usually effective. If the symptoms are severe and persistent, reduction of small bowel activity must be obtained by other means. Vagotomy has been recommended[87] but has little effect.[43, 48] Very high doses of atropine may be of value[48] although the side effects make this method of treatment unsuitable. Luminal also is of no value[85] but the ganglionic blocking agents, taken about half an hour before meals are effective in about 40% of cases,[31, 70, 84] hexamethonium bromide being apparently more effective than Banthine in some cases.[85] In 2 cases Probanthine succeeded in controlling the symptoms most effectively.

Only if the symptoms are severe and disabling for a year after operation in spite of this conservative management should surgical treatment be contemplated.[4, 32, 35, 43, 71, 82, 92] Three cases in the present series were operated upon. 'Hitching up' of the afferent loop was performed in 2 cases with no improvement, as can be expected from what has been said. A long anastomosis between afferent and efferent loops up to the stomach (pantaloon operation) does not delay gastric emptying but merely enlarges the stomach cavity and it is thus not surprising that it is only moderately effective.[92] The recently described operations in which portions of the colon[61, 62] or jejunum[37] are inserted to take the place of the part of the stomach resected have a significant mortality rate[32] and a high incidence of stomal ulcers.[100] They should not be used in their present form.[43] The best operation is to restore gastro-duodenal continuity by an end-to-side anastomosis of gastric remnant to the second part of the duodenum.[92] This was used in 1 case with gratifying success.

2. MALABSORPTION OF FOOD

The intestinal hurry produced by the rapid gastric emptying also results in a disturbance of absorption of the food eaten. This may manifest as vitamin deficiencies but much more commonly the fat, protein and carbohydrate malabsorption give rise to complications.

Normally the presence of fat in the duodenum delays gastric emptying and consequently fat leaves the normal stomach very slowly; carbohydrate normally leaves the stomach very rapidly[7] and protein has an intermediate position. As can be expected from this, carbohydrate, which normally leaves the stomach very rapidly, will be absorbed without much difficulty after the rapid gastric emptying following gastrectomy, whereas fat, which normally enters the bowel slowly, will not be absorbed well if it enters the bowel precipitately. Protein fills an intermediate position between these two.

It is thus not unexpected to find that 63% of patients after gastrectomy have defective fat absorption, although only a few manifest as clinical cases of steatorrhoea.[40, 47, 75] As the condition is dependent on rapid gastric emptying with resultant bowel hypermobility, it is more common after the Polya type of operation than the Billroth I operation[20] and as it results in poor fat absorption it is an important cause of post-operative loss of weight.[11, 105]

In such cases some authors recommend a low fat intake,[15] but this will seriously impair nutrition. It should be treated by frequent small fat-containing meals,[11, 102] on the basis that it is the disturbance of nutrition which is important and not the bulky pale, fatty, frequent stools. Calcium lactate 10 gr. before meals may help by precipitating the fatty acid

crystals[102] and reducing bowel propulsion by means of ganglionic blocking agents may be effective,[31, 40, 102] but it must be kept in mind that pancreatic secretion will be reduced by these drugs, thus possibly aggravating the condition.[40] Two cases of steatorrhoea have been encountered and both improved considerably on small frequent fat-containing meals and ganglionic blocking agents—in both the loose bulky stools persisted (although better than before the treatment) but their state of nutrition improved tremendously.

Protein absorption is less efficient than carbohydrate but better than fat and consequently balance studies show that only 25% of patients have a defective protein absorption following gastrectomy.[40] Clinical evidence of this is rarely encountered.

Carbohydrate customarily leaves the stomach rapidly and the bowel is consequently able to absorb it quickly. The result of the rapid gastric emptying after gastrectomy is thus that the carbohydrate of the meal is absorbed rapidly to produce an abnormally high blood sugar,[2, 30, 79] which can be used as a criterion of the rate of gastric emptying. This hyperglyceamia produces no symptoms, although, being evidence of the rapid emptying of the stomach, it is regularly seen in subjects with dumping symptoms and is absent in those without such symptoms.[85] In a few cases this hyperglycaemia is followed by hypoglycaemia about 2 hours after the meal with the usual symptoms and signs of hypoglycaemia. During this stage the blood sugar is low and the condition readily responds to glucose by mouth.[2, 3]

The exact mechanism of this hypoglycaemia is uncertain. It does not seem to be due to an increased susceptibility to insulin, nor is it an excessive response to hyperglycaemia, as it does not occur after the administration of intravenous glucose.[85] It is apparently a result of the response to the rapid absorption of carbohydrate, either because the high portal carbohydrate content inhibits glycogenolysis and glyconeogenesis, or, more probably, because of a functional hyperinsulinism which was present before the operation[21] in a subclinical form and which has been aggravated by the usually subclinical hyperinsulinism following the gastrectomy.[85]

The diagnosis of this condition is easy, being a post-cibal syndrome occurring a few hours after the meal (unlike the dumping syndrome) and associated with a low blood sugar. It is, however, a very rare condition and only 1 such case has been encountered personally. The treatment of this condition is obviously to take glucose by mouth and the prevention is to take small amounts of glucose at frequent intervals. This treatment and prophylaxis are eminently satisfactory and this condition is consequently not as disturbing as the true dumping syndrome.[16, 63]

II. THE PYLORUS

It has been shown that the function of the pylorus is to prevent reflux of food and bile from the duodenum into the stomach. All types of gastrectomy involve the resection of the pylorus and consequently biliary reflux into the stomach occurs invariably, irrespective of the type of gastrectomy performed. As the gastric remnant empties rapidly, the stomach is already empty by the time the bile enters it and the gastric mucosa is now subjected to the effect of pure, undiluted bile.

This has 2 effects:

1. It produces nausea and vomiting shortly after eating, the vomitus consisting of a small volume of bile without food. The small volume of the bile vomited differentiates it from afferent loop obstruction. This is very common, occuring after 8—10% of gastrectomies (of all types)[43] and 8 cases have been seen in this series—6 after a Polya type of operation and 2 after a Billroth I gastrectomy. The occurrence after Billroth I operations is also unlike afferent loop obstruction. The use of a valve does, to a certain extent, direct the bile into the efferent loop away from the stomach and so tends to decrease this complication. It is also less severe and less persistent after the Billroth I operation.[56]

This effect of gastrectomy fortunately is not disabling and with reassurance and explanation causes only very slight annoyance. At times it is more severe, however, and 1 patient only stopped vomiting with the use of a milk drip to dilute the biliary reflux. In addition, all these cases improve with time,[57] as if the stomach becomes accustomed to the presence of bile.

Rarely reassurance and time do not produce improvement and then it is wise to exclude the presence of a hiatus hernia and stomal obstruction before proceeding with the treatment.[92] If the condition is very severe and disabling, operative relief may be considered as it may even produce a reflux oesophagitis.[43] Jejunoplasty or entero-anastomosis do not effect a cure because reflux of bile can still occur, and in this way also it can be differentiated from afferent loop obstruction. The

only sure relief is obtained by changing the anastomosis to a Roux-Y type with the afferent loop implanted at least a foot below the stoma. In that case a vagotomy must be added to prevent stomal ulceration.[92, 100]

2. The bile in the stomach produces an irritation of the mucosa which results in a severe erosive gastritis with many small ulcers.[56] This is a very frequent cause of persistent epigastric pain following the operation and these patients respond very unsatisfactorily to medical treatment. They are consequently often diagnosed as stomal ulceration and the diagnosis can only be made at gastroscopy. This has been seen in 2 cases in this series. The numerous small ulcers have a tendency to bleed (as was the case in 1 of these 2 cases) and this may be severe, or slow but persistent, forming the site of blood loss responsible for many cases of post-gastrectomy hypochromic anaemia.

This reflux gastritis may occur after any operation which interferes with the pylorus. Pyloroplasty leaves some sort of functioning pylorus, so this condition is rare after that operation[5, 81] (a point in favour of doing a pyloroplasty with vagotomy). It occurs after many gastro-enterostomies,[80] disappearing once the gastro-enterostomy stoma is closed.[81] After certain gastro-enterostomies a rhythmical pylorus-like activity may be seen at the stoma and these cases do not show this gastritis.[80] One wonders whether this is not the explanation of the better results claimed for gastro-jejunostomy performed at the so-called mid-gastric sphincter.

III. THE DUODENUM

Only the Polya type of operations short-circuit the duodenum and therefore the effects to be mentioned do not occur after Billroth I operations. This is another important reason why the Billroth I operation is preferable whenever possible. The disturbance of nutrition is less[105] and normal weight is better maintained.[58, 73]

The effects of short-circuiting the duodenum are:

1. Normally the presence of food and hydrochloric acid in the duodenum inhibit gastric emptying, both reflexly and by the secretion of enterogastrone,[27] and the loss of this inhibition will accentuate the rapid emptying of the stomach with all its effects.[102]

2. The presence of food in the duodenum stimulates the secretion of secretin and pancreazymin which control pancreatic secretion. The loss of these factors results in poor pancreatic secretion which accentuates the poor fat digestion with resultant steatorrhoea and loss of weight.[100] Pancreatin in divided doses of 2.5 g. before, during and after each meal has been recommended,[40] but when used in 1 patient with a feeding jejunostomy it had no significant effect. Calcium lactate 10 gr. before meals has been suggested, to precipitate the fatty acid crystals in the bowel, but is of doubtful value.

3. Food in the duodenum stimulates the secretion of cholecystokinin which is largely responsible for the discharge of bile into the duodenum at the appropriate time. The loss of this function will also impair fat digestion with resultant steatorrhoea and loss of weight. In these cases bile salts (8 g. per day in divided doses) are claimed to be of value but this was not the experience in the case with the jejunostomy. An emulsifying agent, Tween 80, in doses of 1.5 g. 3 times per day has been proposed therapeutically.

4. Such biliary and pancreatic secretion which does take place cannot mix with the food but follows it down the bowel and thus proper mixing does not occur, with a resultant poor digestion.[100] Apart from the methods already mentioned, it may be worth trying a small meal before the main meal, to stimulate biliary and pancreatic secretions which can then be in the jejunum when the food enters the bowel.

5. Duodenal secretion plays a significant role in rendering the gastric chyme isotonic[27] and the loss of this function is an additional cause for the hypertonic chyme entering the jejunum, thus aggravating the 'dumping syndrome'.

6. The duodenum, being a stagnant loop without hydrochloric acid to keep it sterile, may become infected with bacteria not normally found there, to produce an infective enteritis with diarrhoea, steatorrhoea, anaemia and vitamin deficiencies. The management of this condition may be very difficult and restoration of gastro-duodenal continuity may be required.

In conclusion it must again be stressed that, although this list of side effects may look formidable, in actual fact serious symptoms are rare and therefore the operation must still be performed, provided the indication for the procedure is a good one. After the operation the new gastric function should be explained to the patient to ensure a good dietary intake. Any minor post-cibal symptoms which may arise will then be accepted readily in exchange

for the original symptoms. If significant effects do occur they should be treated vigorously, in which case a satisfactory response is almost invariable.

SUMMARY

A collected series of 61 cases with post-gastrectomy symptoms is reviewed.

After excluding unassociated diseases and recurrences of the original disease, the true post-gastrectomy symptoms may be subdivided into mechanical and physiological complications.

The mechanical effects of afferent loop obstruction, afferent loop filling and efferent loop obstruction are due to surgical errors, can be diagnosed readily and should be treated surgically.

The physiological disturbances depend on the structural alteration of the stomach, pylorus and duodenum.

The gastric effects include disturbances of the reservoir function, gastric secretions of intrinsic factor and hydrochloric acid and controlled gastric emptying.

The mechanism of gastric emptying is considered, including the effect of gastrectomy which results in rapid emptying with the dumping syndromes and impaired absorption of food eaten.

The pylorus does not control gastric emptying but prevents duodenal reflux. After gastrectomy this occurs and produces biliary vomiting and erosive gastritis.

The duodenum is short-circuited in the Polya type of gastrectomy. This has many adverse effects on digestion.

The prevention and management of all these conditions are considered.

These complications are rare and do not contra-indicate the operation, provided it is performed for a good reason.

OPSOMMING

'n Reeks van 61 gevalle van na-gastrektomie-simptome word in oënskou geneem.

Na uitsluiting van nie-verwante siektes en nuwe invalle van die oorspronklike siekte kan die suiwere na-gastrektomie-simptome in meganiese en fisiologiese komplikasies onderverdeel word.

Die meganiese effek van aanvoerende boogobstruksie, aanvoerende boogvulling en afvoerende boogobstruksie is te wyte aan chirurgiese vergissinge. Hulle kan maklik gediagnoseer, en behoort chirurgies behandel te word.

Die fisiologiese versteurings, daarenteen, word veroorsaak deur die struktuurverandering van die maag, die maaguitgang en die twaalfvingerige derm.

Die maageffekte behels versteurings van die reservoirfunksie, maagafskeiding van die intrinsieke faktor en soutsuur, en gekontroleerde maagontlediging.

Die meganisme van maagontlediging word oorweeg, asook die effek van gastrektomie wat uitloop op vinnige ontlediging met die dumping-sindrome en versteurde opneming van die voedsel wat geëet word.

Die maaguitgang kontroleer nie maagontlediging nie, maar voorkom duodenale terugvloeiing. Dit vind plaas na gastrektomie, en loop uit op galbraking en verwerende maagontsteking.

In gastrektomie van die Polya-tipe word die twaalfvingerige derm kortgesluit. Dit bring talle nadelige gevolge vir die spysvertering mee.

Die voorkoming van en beheer oor al hierdie toestande word oorweeg.

Hierdie verwikkelinge is seldsame verskynsels en moet nie beskou word as 'n kontra-indikasie vir die operasie nie—mits dit om goeie en voldoende redes uitgevoer word.

I acknowledge with pleasure the assistance I have had from many physicians and surgeons who have given me access to their patients, and to the Superintendent of the Groote Schuur Hospital for permission to publish reports of the cases, most of which were treated in that hospital. A special word of thanks is due to Dr. A. D. Keet of the Groote Schuur Hospital for his unfailing co-operation, and for the films of the solid barium meal illustrated.

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ANNOTATION

PROGNOSIS AND TREATMENT OF CORONARY THROMBOSIS

It was not so very long ago that a diagnosis of coronary thrombosis was tantamount to a death sentence. Yet even in 1933 Lewis[1] was able to say: 'Originally regarded as a hopeless malady, further observation, especially more accurate diagnosis of the milder cases . . . has lightened the gloom of the previous outlook'. He mentioned cases returning to live on in comparative activity for periods of up to 10 and occasionally 15 years or more. In 1942 he added 'or even 20 years' to the last sentence. It is not surprising, therefore, to find practitioners mentioning survival of patients for over 25 years[2] and, especially in view of the more recent advances in diagnosis and treatment, justifiably adopting a more hopeful outlook.

Diagnosis has improved greatly with the free use of the multi-lead electrocardiogram. Many mild cases are recognized and the previous clear distinction between angina of effort on the one hand and coronary thrombosis on the other has disappeared with the recognition of all grades of 'coronary insufficiency'. Recognition of these and of atypical cases previously unrecognized as cases of coronary infarction has enabled treatment to be instituted early and in some cases has prevented unnecessary and dangerous surgery.

In the acute attack rest and sedation still remain the sheet anchor of therapy. The ill effects of ambulation have long been appreciated and have recently been re-emphasized.[3] Anticoagulant therapy is widely used and has been discussed in these columns.[4] The treatment of shock in the acute phase has received more consideration in recent years. Four out of 5 patients with grave shock die. Intravenous infusions of hypertonic glucose, plasma, blood and dextran have been used with little success, but better results have been claimed with *l*-noradrenalin, though the mortality is still considerable.[5] Mild cases appear to do well if treated with rest alone.[6]

The long-term outlook in this disease requires re-orientation. It has been shown that some evidence of coronary disease was present in 77.3% of young soldiers killed in action in Korea.[7] The average age of this group of apparently fit men was 22.1 years. Even if clinically important coronary artery disease was present in only 22% of these cases,[8] it seems clear that the disease is widespread. It probably needs to be far advanced —or very strategically situated—before symptoms or signs occur. The clinical occurrence of the disease in young adults is receiving more recognition. Gertler and White[9] have extended the work of Glendy, Levine and White.[10] They analysed 100 patients (under the age of 40 years) who had clinical coronary artery disease and compared them with a series of matched controls. In this group of apparently 'pure' coronary heart disease (i.e. uncomplicated by hypertension, xanthomatosis, diabetes or nephrosis) there were 97 males and only 3 females. Of the 21 men who died while the study was in progress, the average survival period following the initial coronary episode was 6½ years with a range from 1½ to 12¼ years. Of the men still alive at this date, the average survival was 8 7/12 years with a range from 4 7/12 to 26¾ years.

What of long term therapy? Surgical procedures have been tried. Some success has been claimed with procedures varying from omentopexy, installation of talc into the pericardial cavity,[11] implantation of an artery into the cardiac muscle[12] and arterialization of the coronary sinus.[13] In view of the type of patient operated on, immediate mortality has been relatively low but clearly these procedures cannot as yet be used widely. Palliative surgery, e.g. sympathectomy for relief of pain, has a place in carefully selected cases as have measures which produce myxoedema.

A more hopeful approach to the problem is the metabolic or endocrine one. In this disease there is some alteration of the serum cholesterol : phospholipid ratio. Whether this is the primary defect or whether it is a manifestation of a wider one is still in doubt. A low fat diet can lower serum cholesterol (though after a time the level appears to rise again) and it is possible that a fat-poor diet may improve the long-term outlook. It does not seem to matter if fat is ingested as fat or as cholesterol; nor is it certain whether coronary atheroma with deposition of cholesterol in the coronary artery is the primary lesion. Duguid[14] believes that thrombosis is the fundamental cause of coronary artery narrowing and it is the clot, later incorporated

into the vessel wall, which is the cause of the 'atheromatous patch'. If this is so, then it may provide a rational basis for long-term anticoagulant therapy which has been claimed to lower mortality.

Oestrogen therapy has lately been tried mainly because of the rarity of clinical coronary sclerosis in females before the menopause. Furthermore, in men treated with oestrogens for carcinoma of the prostrate, atherosclerotic lesions appeared to be comparatively sparse. In men with coronary sclerosis the plasma ester and total cholesterol, the total cholesterol/phospholipid ratio and the concentration of cholesterol and the β-lipoprotein fraction, were strikingly depressed by ethinyl oestradiol. Toxic effects were troublesome and difficult to eliminate.[15] Nevertheless, this type of treatment also offers hope for the future.

Finally, one could consider a direct attack on the coronary arteries themselves. Visualization of the coronary tree with radio-opaque substances, isolation and removal of narrowed segments and their replacement with grafts of some kind become possible with the newer techniques of cardiac surgery. Alternatively one could 'dissolve' the clots in the lumen of the vessels. Anticoagulants as presently used do not seem to accomplish this. It is true that removal of artificially produced thrombi in the limb vessels of rabbits with Tromexan is possible.[16] Fibrinolytic substances may be more successful,[17, 18] and it may eventually be possible to open thrombosed vessels by their use. Perhaps (at the age of say 20 years for men and 40 years for women) all of us may enter hospital for prophylactic 'fibrinolysis'—a sort of annual 'rebore'!

OPSOMMING

Die gebruik van die elektrokardiograaf met meer as een geleiding het 'n groot verbetering in die diagnose van koronêre trombose tot gevolg gehad. Talle ligte gevalle word tans herken, en die vroeëre duidelike onderskeid tussen die angina wat op kragsinspanning volg en koronêre trombose het verdwyn met die herkenning van alle soorte koronêre gebreke. Daarbenewens het die herkenning van atipiese gevalle wat vroeër nie as infarkt-gevalle beskou is nie, dit moontlik gemaak om die behandeling vroeër 'n aanvang te laat neem.

In akute aanvalle is rus en kalmering nog steeds die grondslag van die terapie. Ander aspekte van die behandeling word in oënskou geneem, en die noodsaaklikheid aan 'n reorientasie van die langtermyn-kyk op hierdie siekte word beklemtoon.

'n Hoopvoller benadering word deur die metaboliese en endokrien-studies aangedui.

'n Regstreekse aanval op die koronêre slagare self is nie heeltemal buite die kwessie nie. Later sal dit miskien moontlik wees om vate waarin 'n klont gevorm is, oop te maak deur die gebruik van fibrinolitiese stowwe.

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MEDIES-GEREGTELIKE AFDELING · MEDICO-LEGAL SECTION

WET OP GENEESHERE, TANDARTSE EN APTEKERS

VORDERINGS DEUR ANDER GEREGISTREERDE PERSONE AS APTEKERS

80*bis*. (1) Tensy die omstandighede dit vir hom onmoontlik maak, moet elke ingevolge hierdie Wet geregistreerde persoon (in hierdie artikel die prak-

MEDICAL, DENTAL AND PHARMACY ACT

CHARGES BY REGISTERED PERSONS OTHER THAN CHEMISTS AND DRUGGISTS

80*bis*. (1) Every person registered under this Act (in this section referred to as the practitioner) except a chemist and druggist shall, unless the circum-

tisyn genoem), behalwe 'n apteker, voordat hy professionele dienste lewer, die persoon aan wie die dienste gelewer gaan word of iemand wat vir die onderhoud van daardie persoon verantwoordelik is, verwittig van die gelde wat hy voornemens is om vir daardie dienste te vorder—

(*a*) wanneer daartoe deur die betrokke persoon versoek; of

(*b*) wanneer daardie gelde meer is as die gelde wat gewoonlik vir sulke dienste gevorder word, en moet in 'n geval waarop paragraaf (*b*) betrekking het, die betrokke persoon ook van die gewone gelde verwittig.

(2) 'n Praktisyn wat ten opsigte van professionele dienste deur hom gelewer betaling van iemand (in hierdie artikel die pasiënt genoem) vorder, moet binne veertien dae na ontvangs van 'n skriftelike versoek te dien effekte aan die pasiënt 'n gespesifiseerde rekening en die verdere inligting wat die pasiënt mag verlang met betrekking tot die gevorderde bedrag verstrek.

(3) Die pasiënt kan binne veertien dae na ontvangs van die in sub-artikel (2) bedoelde gespesifiseerde rekening en verdere inligting, as daar is, die praktisyn skriftelik meedeel dat die gevorderde bedrag na sy oordeel onredelik is en die gronde waarop sy sienswyse berus, uiteensit en moet terselfdertyd besonderhede van die vordering en van die bedoelde gronde aan die raad deurstuur.

(4) Die praktisyn kan binne veertien dae na ontvangs van die in sub-artikel (3) bedoelde gronde aan die pasiënt 'n gewysigde vordering ter vervanging van sy oorspronklike vordering voorlê en moet indien hy aldus 'n gewysigde vordering voorlê, 'n afskrif daarvan aan die raad deurstuur.

(5) Indien die pasiënt geen antwoord van die praktisyn ontvang nie en geen gewysigde vordering voorgelê word nie, of indien 'n gewysigde vordering voorgelê word en die pasiënt die gewysigde vordering ook as onredelik beskou, kan die pasiënt binne veertien dae nadat die in sub-artikel (4) bedoelde tydperk verstryk het of na ontvangs van die gewysigde vordering, by die raad aansoek doen om 'n vasstelling van die maksimum bedrag wat die praktisyn ten opsigte van die gelewerde dienste van die pasiënt moes gevorder het.

(6) 'n Aansoek ingevolge sub-artikel (5) gaan vergesel van—

(*a*) 'n beëdigde verklaring wat volledig uiteensit—

(i) die gronde waarop die pasiënt se sienswyse dat die gevorderde bedrag onredelik is, berus; en

(ii) die feite met betrekking tot die gevorderde bedrag wat die pasiënt onder die aandag van die raad wil bring; en

(*b*) 'n vasstellingsgeld van vyf persent van die gevorderde bedrag.

(7) Die pasiënt moet op die datum waarop hy 'n aansoek ingevolge sub-artikel (5) aan die raad deurstuur, per aangetekende pos 'n afskrif van die in paragraaf (*a*) van sub-artikel (6) bedoelde beëdigde verklaring aan die praktisyn stuur, en die praktisyn kan binne veertien dae na ontvangs daarvan aan die raad 'n beëdigde verklaring voorlê wat volledig uiteensit—

(*a*) enige vertoë wat hy ter ondersteuning van sy vordering wens voor te lê; en

(*b*) die feite met betrekking tot die gevorderde bedrag wat hy onder die aandag van die raad wil bring.

(8) By ontvangs van 'n aansoek ingevolge sub-artikel (5) benoem die raad minstens twee en hoogstens vyf van sy lede as assessors wat die maksimum bedrag vasstel wat die praktisyn na hulle oordeel ten opsigte van die gelewerde dienste van die

stances render it impossible for him to do so, before rendering any professional services inform the person to whom the services are to be rendered or any person responsible for the maintenance of such person, of the fee which he intends to charge for such services—

(*a*) when so requested by the person concerned; or

(*b*) when such fee exceeds that usually charged for such services, and shall in a case to which paragraph (*b*) relates also inform the person concerned of the usual fee.

(2) Any practitioner who in respect of any professional services rendered by him claims payment from any person (in this section referred to as the patient) shall, within fourteen days after receipt of a request in writing to that effect provide the patient with a detailed account and such further information relating to the amount claimed as the patient may require.

(3) The patient may within fourteen days after receipt of the detailed account and further information, if any, referred to in sub-section (2), in writing inform the practitioner that in his opinion the amount claimed is unreasonable and set out the grounds on which such opinion is based and shall at the same time transmit to the council particulars of the claim and of the said grounds.

(4) The practitioner may within fourteen days after receipt of the grounds referred to in sub-section (3), submit to the patient an amended claim in substitution for his original claim and shall if he so submits an amended claim transmit to the council a copy thereof.

(5) If no reply is received by the patient from the practitioner and no amended claim is submitted, or if an amended claim is submitted and the patient considers such amended claim also to be unreasonable, the patient may, within fourteen days after the expiration of the period referred to in sub-section (4) or after receipt of the amended claim, apply to the council for a determination of the maximum amount which the practitioner should have claimed from the patient in respect of the services rendered.

(6) An application under sub-section (5) shall be accompanied by—

(*a*) an affidavit setting forth fully—

(i) the grounds upon which the patient's opinion that the amount claimed is unreasonable, is based; and

(ii) such facts relating to the amount claimed as the patient may wish to bring to the notice of the council; and

(*b*) a determination fee of five per cent. of the amount claimed.

(7) The patient shall on the date upon which he transmits to the council an application under sub-section (5), transmit by registered post to the practitioner a copy of the affidavit referred to in paragraph (*a*) of sub-section (6), and the practitioner shall within fourteen days after receipt thereof submit to the council an affidavit setting forth fully—

(*a*) any representations he may wish to submit in support of his claim; and

(*b*) such facts relating to the amount claimed as he may wish to bring to the notice of the council.

(8) Upon receipt of an application under sub-section (5), the council shall appoint not less than two and not more than five of its members as assessors who shall determine the maximum amount which in their opinion the practitioner should have claimed from the patient in respect of the services rendered.

(9) The assessors shall for the purpose of making

pasiënt moes gevorder het.

(9) Die assessors moet vir die doel van 'n vasstelling ingevolge sub-artikel (8), aan sowel die praktisyn as die pasiënt 'n geleentheid bied om by beëdigde verklaring sodanige vertoë in te dien, benewens enige vertoë ingevolge sub-artikel (6) of (7) ingedien, as wat hy vir oorweging deur die assessors wil voorlê.

(10) 'n Vasstelling ingevolge sub-artikel (8) gemaak, word per aangetekende pos aan die praktisyn en die pasiënt meegedeel en is, behoudens die bepalings van sub-artikels (11) en (12), afdoende en bindend vir hulle.

(11) 'n Praktisyn of pasiënt wat hom veronreg voel deur 'n vasstelling ingevolge sub-artikel (8), kan binne dertig dae na die datum daarvan en teen betaling aan die raad van 'n hersieningsgeld van vyf persent van die ingevolge daardie sub-artikel vasgestelde bedrag, by die raad om hersiening van die vasstelling aansoek doen.

(12) By ontvangs van 'n aansoek ingevolge sub-artikel (11) moet die raad of, indien die gevorderde bedrag minder as honderd pond is, sy uitvoerende komitee die vasstelling van die assessors hersien, en kan hy daardie vasstelling bekragtig of dit ter syde stel en opnuut die maksimum bedrag vasstel wat die praktisyn ten opsigte van die gelewerde dienste van die pasiënt moes gevorder het.

(13) Die bepalings van sub-artikels (9) en (10) is *mutatis mutandis* ten opsigte van 'n vasstelling by hersiening kragtens sub-artikel (12) van toepassing.

(14) Indien die ingevolge sub-artikel (8) vasgestelde bedrag minder as negentig persent van die gevorderde bedrag is, en nie by hersiening ingevolge sub-artikel (12) tot negentig persent of meer van die gevorderde bedrag vermeerder word nie, kan die pasiënt 'n bedrag gelyk aan die vasstellingsgeld op die praktisyn verhaal.

(15) Indien die bedrag by hersiening ingevolge sub-artikel (12) vasgestel—

(*a*) minder as negentig persent van die gevorderde bedrag en minder as die tevore ingevolge sub-artikel (8) vasgestelde bedrag is, kan die pasiënt, waar die aansoek om hersiening deur hom gedoen is, 'n bedrag gelyk aan die hersieningsgeld op die praktisyn verhaal;

(*b*) negentig persent of meer van die gevorderde bedrag en meer as die tevore ingevolge sub-artikel (8) vasgestelde bedrag is, kan die praktisyn, waar die aansoek om hersiening deur hom gedoen is, 'n bedrag gelyk aan die hersieningsgeld op die pasiënt verhaal.

(16) Hangende die uitslag van 'n aansoek om 'n vasstelling ingevolge sub-artikel (5) of die hersiening van 'n vasstelling ingevolge sub-artikel (12), word geen geding deur 'n praktisyn vir die verhaal op 'n pasiënt van 'n bedrag wat by so 'n aansoek betrokke is, ingestel nie.

(17) Waar die bedrag wat 'n praktisyn ten opsigte van deur hom gelewerde professionele dienste moes gevorder het, kragtens hierdie artikel vasgestel is, is daardie praktisyn nie geregtig om ten opsigte van daardie dienste 'n hoër bedrag as die aldus vasgestelde bedrag op die pasiënt te verhaal nie.

(18) Hierdie artikel word nie geag die raad van enige van sy bevoegdhede of werksaamhede ingevolge Hoofstuk IV met betrekking tot handelinge of versuime waarvan hy kennis mag neem, te onthef nie.".

[*Uittreksels uit die* Wet op Geneeshere, Tandartse en Aptekers *word herdruk kragtens kopieregmagtiging van die Staatsdrukker Nr.* 2189 *van* 31 *Maart* 1955.—*Redakteur*]

a determination under sub-section (8), afford both the practitioner and the patient an opportunity of submitting by affidavit such representations, in addition to any representations submitted under sub-section (6) or (7), as they may wish to put forward for consideration by the assessors.

(10) Any determination made under sub-section (8) shall be communicated by registered post to the practitioner and the patient and shall, subject to the provisions of sub-section (11) and (12), be final and binding on them.

(11) Any practitioner or patient who considers himself aggrieved by a determination under sub-section (8) may, within thirty days after the date thereof and upon payment to the council of a review fee of five per cent. of the amount determined under that sub-section, apply to the council for a review of the determination.

(12) Upon receipt of an application under sub-section (11), the council or, if the amount claimed is less than one hundred pounds, its executive committee, shall review the determination of the assessors and may confirm that determination or set it aside and determine anew the maximum amount which the practitioner should have claimed from the patient in respect of the services rendered.

(13) The provisions of sub-sections (9) and (10) shall apply *mutatis mutandis* in respect of a determination on review under sub-section (12).

(14) If the amount determined under sub-section (8) is less than ninety per cent. of the amount claimed, and is not on review under sub-section (12) increased to ninety per cent. or more of the amount claimed, the patient may recover from the practitioner an amount equal to the determination fee.

(15) If the amount determined on review under sub-section (12)—

(*a*) is less than ninety per cent. of the amount claimed and less than the amount previously determined under sub-section (8), the patient may, where application for review was made by him, recover from the practitioner an amount equal to the review fee;

(*b*) is ninety per cent. or more of the amount claimed, and exceeds the amount previously determined under sub-section (8), the practitioner may, if the application for review was made by him, recover from the patient an amount equal to the review fee.

(16) Pending the result of an application for a determination under sub-section (5) or the review of a determination under sub-section (12), no proceedings shall be instituted by a practitioner for the recovery from a patient of any amount involved in such application.

(17) Where the amount which should have been claimed by a practitioner in respect of any professional services rendered by him has been determined under this section, that practitioner shall not be entitled to recover from the patient concerned in respect of these services an amount exceeding the amount determined.

(18) This section shall not be deemed to divest the council of any any of its powers or functions under Chapter IV in relation to acts or omissions of which it may take cognizance.

[*Extracts from the* Medical, Dental and Pharmacy Act *reproduced under Government Printers' Copyright Authority No.* 2189 *of* 31 *March* 1955.—*Editor*]

PREPARATE EN TOESTELLE

KORTIKOÏED-PYNSTILLENDE TERAPIE

SIGMAGEN: 'N NUWE SCHERING-MIDDEL

Sigmagen, 'n nuwe kortikoïed-pynstillende preparaat waarin die nuwe ontstekings- en rumatiekbestrydende kortikosteroïed ‚Meti-drug'-prednisoon met asperien, vitamien C en suurbestrydende aluminiumhidroksied verenig is, word deur Scherag (Pty.) Limited, Johannesburg, aangekondig.

Sigmagen, wat ontwikkel is deur die ontdekkers van prednisoon (‚Meticorten') en prednisoloon (‚Meticortelone'), is die rasionele terapeutiese benadering tot die probleem van die behandeling van sulke rumatiek-arthritiskwale soos ligte rumatiekagtige gewrigsontsteking of werwelontsteking, subakute of tussenpoos-jig, slymbeursontsteking, spierontsteking, fibrositis en neuritis. Dit is kwale waaraan miljoene mense ly, maar in die reël word hulle nie so ernstig beskou dat hulle volle steroïed-hormoonbehandeling regverdig nie.

Sigmagen in tabletvorm het die aanvullende effek van die pynstillende middel wat die meeste in die geneeskunde gebruik word, sowel as die effek van die nuwe ‚Meti-steroid' wat 3-5 keer doeltreffender as kortisoon of hidrokortisoon is vir die bestryding van ontsteking en rumatiek.

Kliniese studies het bewys dat, in sommige gevalle, die gelyktydige toediening van die kortikoïede en die salisilate 'n kleiner dosis van elk moontlik maak om dieselfde of 'n selfs beter terapeutiese effek te verkry. Hierdie vermindering van dosis maak onwenslike reaksies ook minder waarskynlik.

Die askorbiensuur-bestanddeel van ‚Sigmagen' help om te voorsien in die toenemende behoefte aan hierdie vitamien tydens ongesteldheid, terwyl die maagkwale wat soms voorkom by pasiënte wat groot dosisse van die salisilate ontvang, verlig of uitgeskakel word deur die aluminiumhidroksied in die ‚Sigmagen'-formule.

Ná behandeling met ‚Sigmagen' bedaar die subjektiewe simptome, soos die styfheid en die pyn wat deur bewegings veroorsaak word. Later word 'n objektiewe verbetering (insluitende 'n vermindering van die grootte van die gewrigte, en toenemende beweeglikheid) waargeneem. 'n Verbetering in die algemene gevoel van welsyn is gewoonlik ook te bespeur.

Dosis en Verpakking: ‚Sigmagen' word beskikbaar gestel in buisies van 12 en in bottels van 100 tablette, en word soos volg toegedien:

In akute toestande 2, of, indien nodig, 3 ‚Sigmagen'-tablette 4 maal per dag totdat bevredigende resultate verkry word, waarna die dosis tot 1 of 2 tablette al om die ander dag verminder word. Later word die behandeling dan gestaak.

In kroniese of subakute gevalle word 1 ‚Sigmagen'-tablet 4 maal per dag voorgeskryf, en die dosis word geleidelik vermeerder tot 3 tablette 4 maal per dag, indien nodig, totdat 'n bevredigende reaksie verkry word. Die behoorlike instandhoudingspeil word dan vasgestel deur die dosis geleidelik te verminder met 1 tablet al om die 2 of 3 dae. Vir die beste resultate moet ‚Sigmagen'-tablette ná maaltye en met slapenstyd geneem word.

'N NUWE VERPAKKING VIR ENTACYL-SWEEFMENGSEL

British Drug Houses kondig aan dat 'n enkele-dosishouer vir die een-dag-behandeling van askariase tans beskikbaar is.

Iedere bottel bevat 28 ml. Entacyl-sweefmengsel, wat voldoende is vir die behandeling van askariase by een volwassene of etlike kinders. Hierdie verpakking is ideaal geskik vir die grootskeepse behandeling van aangetaste gemeenskappe, soos aanbeveel deur drs. Hanna en Shehata in Brit. Med. J., 13 Augustus 1955, **2**, 417. Die aanbevole dosis is 750 mg. piperasien-adipaat (4.4 ml.) per lewensjaar tot 6 jaar, en 4.5 g. piperasien-adipaat (26.6 ml.) vanaf 6 jaar en ouer. Dit word op 'n enkele dag toegedien in 4 gelyke dele by tussenpose van 4 uur ná maaltye.

Afgesien van die besuiniging wat dit in algemene gebruik sal meebring, voel ons oortuig dat hierdie verpakking byval sal vind by geneeshere wat voor die probleem te staan gekom het om askariase uit te roei by 'n groot aantal pasiënte op wie daar nie altyd staatgemaak kan word dat hulle vir behandeling na die dokter sal kom nie.

Leesstof en monsters is op aansoek verkrybaar.

'N NUWE SOORT TABLETBEDEKKING

(ABBOTT LABORATORIES)

'n Nuwe tabletbedekking wat vinnig, volkome en op 'n betroubare wyse oplos, word tans gebruik vir 'n hele paar van die produkte van Abbott Laboratories. Hierdie nuwe bedekking wat Filmtab genoem word, werk vinniger opneming van die geneesmiddel en 'n vroeër, doeltreffende bloedpeil in die hand. Die voordele daarvan is op 'n dramatiese wyse aangetoon in die geval van Abbott se Erythrocin-Filmtab. Met 'n Filmtab-bedekking gee Erythrocin nou 'n terapeutiese bloedpeil binne minder as 2 uur—in plaas van die gewone 4-6 uur.

Filmtab self is 'n weefseldun bedekking—so dun dat opbreking 'n aanvang neem binne 30 sekondes na die inneming van die tablet. Algehele oplossing geskied binne 5-10 minute.

Die dunheid van Filmtab het 'n kleiner tablet wat makliker is om in te sluk, moontlik gemaak. Ook die tabletbeskerming is groter. Filmtab word nie sag of klewerig nie, met die gevolg dat die tablette nie aan mekaar vassit nie. Filmtab sal ook nie uitdroog of bars nie. 'n Verdere voordeel is dat Filmtab volkome smaakloos is, en die smaak van die tablet self vermom.

Hierdie eksklusiewe tabletbedekking word tans onder meer vir die volgende Abbott-produkte gebruik: Dicalets Filmtabs, Di-Paralene-Hidrochloried-Filmtabs, Erythrocin-Stearaat-Filmtabs en Iberol Filmtabs.

PREPARATIONS AND APPLIANCES

CORTICOID ANALGESIC THERAPY

SIGMAGEN: A NEW SCHERING DRUG

Sigmagen, a new corticoid-analgesic preparation combining the new anti-inflammatory-antirheumatic corticosteroid 'Meti-drug' prednisone with aspirin, vitamin C, and antacid aluminium hydroxide has been announced by Scherag (Pty.) Limited, Johannesburg.

Developed by the discovers of prednisone ('Meticorten') and prednisolone ('Meticortelone'), Sigmagen provides a rational therapeutic approach to the treatment of such rheumatic-arthritic disorders as mild rheumatoid arthritis or spondylitis, subacute or interval gout, bursitis, myositis, fibrositis and neuritis. These are conditions which afflict millions of people but which are not generally considered to indicate full steroid hormone treatment.

In tablet form, Sigmagen exerts the complementary action of medicine's most widely used analgesic and the new 'Meti-steroid' that has 3-5 times the antirheumatic and anti-inflammatory effectiveness of cortisone or hydrocortisone.

Clinical studies indicate that in some cases the concomitant administration of corticoids and salicylates permits a lower dosage of each to achieve an equal or better degree of therapeutic effect. This decrease in dosage further reduces the likelihood of undesirable effects.

The ascorbic acid component in 'Sigmagen' helps meet the increased daily need for this vitamin during stressful conditions, while gastric distress, sometimes occurring in patients receiving large doses of salicylates, is relieved or prevented by the aluminium hydroxide included in the 'Sigmagen' formula.

Following treatment with 'Sigmagen', subjective symptoms (such as aching, stiffness and pain on motion) subside first, with objective improvement (including decrease in joint size and increased motion) noted later. An improvement in the general sense of well-being is usually observed.

Dosage and Packaging: 'Sigmagen' is supplied in tubes of 12 and bottles of 100 tablets and is administered as follows:

In acute conditions—2, or when necessary, 3 'Sigmagen' tablets 4 times daily until a satisfactory result is obtained, after which the dosage is reduced 1 or 2 tablets every other day and then discontinued.

In chronic or subacute conditions 1 'Sigmagen' tablet 4 times daily and the dosage is increased gradually to 3 tablets 4 times daily, if required, to achieve a satisfactory response. The proper maintenance level may then be determined by gradually reducing the dosage 1 tablet every 2 or 3 days. For optimal results, 'Sigmagen' tablets should be administered after meals and at bedtime.

NEW PACKING OF ENTACYL SUSPENSION

British Drug Houses announce that they now have available a single dose container for the one-day treatment of ascariasis.

Each bottle contains 28 ml. of Entacyl Suspension sufficient for the treatment of ascariasis in one adult or several children. This packing is ideally suited for the mass treatment of affected communities, as recommended by Drs. Hanna and Shehata in the Brit. Med. J. of 13 August 1955, **2**, 417. The recommended dose is 750 mg. of piperazine adipate (4.4 ml.) per year of life up to 6 years and 4.5 g. of piperazine adipate (26.6 ml.) from 6 years upwards to be given on one day in 4 equal parts at 4-hourly intervals after meals.

Apart from its economy in general usage, this packing will appeal to those doctors faced with the problem of eradication of ascariasis in a large number of patients whose attendances for treatment are unreliable.

Literature and samples are available on request.

A NEW TYPE OF TABLET COATING

(ABBOTT LABORATORIES)

A new tablet coating which dissolves quickly, completely and dependably is currently being introduced on several products by Abbott Laboratories. Called Filmtab, this new coating permits faster drug absorption and earlier effective blood levels. Its advantage is dramatically shown in the case of Abbott's Erythrocin Filmtab. With Filmtab coating, Erythrocin now gives therapeutic blood levels in less than 2 hours, instead of the usual 4-6 hours.

Filmtab itself is a tissue thin coating—so thin that disintegration starts 30 seconds after tablet ingestion, and takes just 5-10 minutes for complete dissolution.

Filmtab thinness also permits a smaller, easier-to-swallow tablet. Tablet protection is greater, too. Filmtab does not get soft or gummy, so tablets cannot stick together; nor will Filmtab dry out and split. Another advantage is that Filmtab is completely tasteless and also masks taste of the tablet.

Some of the Abbott products now featuring this exclusive tablet coating are Dicalets Filmtabs, Di-Paralene Hydrochloride Filmtabs, Erythrocin Stearate Filmtabs and Iberol Filmtabs.

NOTES AND NEWS · BERIGTE

Dr. R. B. Peckham is now in practice at 139 Loop Street, Pietermaritzburg, Natal.

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Dr. J. J. van der Wat, formerly of Heidelberg, Transvaal, is now resident at 10 Lystanwold Road, Saxonwold, Johannesburg.

Dr. A. D. Bensusan (President of the Photographic Society of Southern Africa) is the first South African to have been honoured by a special service award for 'outstanding service in the promotion of international goodwill in the furtherance of friendly relations among photographers of South Africa and Northern America'.

The award was given by the Photographic Society of America.

* * *

Dr. O. Budtz-Olsen is leaving the Department of Physiology, University of Cape Town, in March to assume a Senior Lectureship in Physiology at the University of Queensland, Brisbane.

* * *

Dr. Norman Sapeika, of the Department of Physiology and Pharmacology, University of Cape Town, has been appointed an Associate Professor.

* * *

Dr. Christine Gilbert has been accorded the honour of an appointment as a Reader in Embryology in the Department of Anatomy in the University of the Witwatersrand.

PROFESSOR WILLIAM W. FRYE

Professor William W. Frye (Dean and Professor of Tropical Medicine, Lousiana State University School of Medicine, New Orleans, U.S.A.) recently visited the Union for 4 weeks on a Research Fellowship provided by the Overseas Division of Pfizer International Medical Service.

Professor Frye (left) in conversation with Dr. R. Elsdon-Dew, head of the C.S.I.R. Amoebiasis Research Unit, Durban, where Professor Frye recently carried out a programme of clinical as well as pathological studies in tropical diseases.

Professor Frye was a member of the U.S. Cholera Commission to China in 1945 and served on the U.S. Commission on Enteric Infections in Korea in 1951. He is now also Special Consultant to the U.S. Public Health Service and the Surgeon-General of the U.S. Army in the field of tropical medicine. He is Chairman of the Combined Deans Committee, Veterans Administration Hospital in New Orleans, and Chairman of the Parasitology and Tropical Medicine Study Section, Research Grants Division, U.S. Public Health Service. Since 1950 he has been Deputy-Director, Commission on Enteric Infections, U.S. Armed Forces Epidemiological Board.

Whilst interested in tropical diseases in general, Professor Frye is specifically concerned with the treatment and control of amoebiasis in various parts of the world. In the U.S.A. very few fulminating cases of amoebiasis are encountered, whereas in Durban this type of case appears frequently, thus providing him with ample material for study.

Professor Frye was particularly interested in the following questions, which he investigated during his visit to South Africa:

(*a*) Why does an apparently dormant case suddenly flare up and become fulminant?

(*b*) Is there any specific nutritional reason for fulminant cases?

(*c*) What changes are required to cause trophozoites suddenly to assume invasive characteristics?

(*d*) What predisposing factors cause fulminant cases in patients taking up residence in Durban?

Professor Frye was also interested in diagnostic methods, since those currently available seem to have severe limitations.

After completing his investigations in the Union, Professor Frye left for India, Pakistan, Malaya, Australia, New Zealand and Japan, in which countries he will continue his study of tropical diseases as part of the programme supported by the Pfizer Fellowship.

* * *

Dr. G. W. van Selm, B.Sc.(S.A.), M.B., B.Ch. (Rand), D.M.R. (Pretoria), has joined Drs. Loots, Osler and Esterhuizen in radiological practice at 101 Medical Centre, 209 Jeppe Street, Johannesburg. (Telephones. *Rooms*: 23-7144/5/6; Residence: 43-3743.)

E. AND S. LIVINGSTONE LTD.: MEDICAL BOOKS CATALOGUE

A complete catalogue of medical books published by E. & S. Livingstone Limited, of 15-17 Teviot Place, Edinburgh 1, Scotland, has just been issued.

Practitioners who are interested in receiving a copy of this comprehensive catalogue are invited to write direct to the publishers at the foregoing address.

BLOCK THAT METAPHOR!

From *A Text-book of Pathology* by William Boyd

It would indeed be rash for a mere pathologist to venture forth on the uncharted sea of the endocrines, strewn as it is with the wrecks of shattered hypotheses, where even the most wary mariner may easily lose his way as he seeks to steer his bark amid the glandular temptations whose siren voices have proved the downfall of many who have gone before.

EMERGENCY WIRELESS CALL SYSTEM FOR MEDICAL PRACTITIONERS IN JOHANNESBURG

Protea Holdings Limited, P.O. Box 7793, Johannesburg (*telephone*: 33-2211), propose to instal a central wireless transmitter, in the region of Hospital Hill, so as to make available an emergency call system for medical practitioners in their motor-cars.

The doctor's car would be fitted with a two-way wireless receiver-transmitter. Should a call come for the practitioner while he is travelling, he could be reached at once by means of this broadcast system.

Provided a certain minimum number of doctors

is interested in this scheme, Protea Holdings Limited would undertake to establish this rapid means of communication.

Those interested should communicate with Protea Holdings Limited at the address given.

MARKED DECLINE IN POLIOMYELITIS IN THE U.S.A.

THE SALK VACCINE

U.S. Government figures show that the weekly incidence of poliomyelitis in 14 states last summer was well below the lowest figures for the previous 5 years. The trend began in the second month after inoculation of more than 2 million children with the Salk vaccine. This was about the time when the first dose normally would produce its protective effect.

In 14 states (Indiana, Alabama, Florida, Georgia, Mississippi, North Carolina, South Carolina, Tennessee, Arkansas, Louisiana, Oklahoma, Texas, Virginia, and West Virginia) only 5% of the total population was inoculated, and only one or two doses were given instead of the 3 required for the full immunization procedure; but the children in the age group inoculated normally have the highest incidence of poliomyelitis.

The Polio Surveillance Unit of the U.S. Public Health Service made a study of 5 million children in 11 scattered states and reported tentative findings that the attack rates for paralytic poliomyelitis in the vaccinated group were from ¼–½ of the rates for the unvaccinated children. Thus the vaccine's effectiveness in reducing paralytic poliomyelitis ranged from 50% to 80%. The average was 76%.

In a special report on New York, the Surveillance Unit disclosed that the paralytic attack rate among children who got 2 doses of vaccine in 1955 was only 3 per 100,000 as compared with 5 per 100.000 for those receiving one dose and 20.9 per 100,000 among the unvaccinated.

State health authorities credit the vaccine for reducing both the incidence and severity of poliomyelitis.

In Louisiana, Dr. S. J. Philips, president of the State Board of Health, said the vaccine caused a 33% decrease in poliomyelitis cases.

The Texas State Department of Health reports that up to 30 September the attack rate for paralytic poliomyelitis among the 473,830 vaccinated children was 1.9 per 100,000 as contrasted with 17.7 per 100,000 among the 378,930 unvaccinated children in the 5-9 age group.

Of the vaccinated children, only 33,000 received the 3 inoculations required in the full immunization procedure. The remaining 440,000 got at least one but not more than 2 doses. The morbidity report concludes: 'On the basis of these figures, it appears that the vaccine was approximately 90% effective in preventing the paralytic form of the disease in Texas.'

In some Northern states, official figures told a similar story. Dr. Herman N. Bundesen, president of the Chicago Board of Health, said the incidence of poliomyelitis among Chicago schoolchildren who received Salk vaccine was cut nearly 90%. The poliomyelitis rate for unvaccinated children was 89.9 per 100,000 compared with 9.3 per 100,000 for those vaccinated.

At the same time, the Salk vaccine reduced the severity of the cases. The rate for paralytic poliomyelitis in the unvaccinated group was 16 times as great as for those who received the vaccine.

In Connecticut, Dr. James C. Hart, Director of the Bureau of Preventable Diseases, disclosed that the case rate for paralytic poliomyelitis among the vaccinated group was 4.7 per 100,000 as compared with 20.1 per 100,000 for the unvaccinated group. Paralytic cases among the vaccinated were milder. Dr. Hart emphasized that the statistics on the 106,120 vaccinated children in Connecticut involved a period during which most had had only one dose of the vaccine. 'In no instance where children received 2 shots and the second had had time to work has there been a single instance of polio of any sort so far,' Dr. Hart said. 'We hope this continues to be a true picture. If it does, it means that one shot has done half the job and the second has acted as a strong booster.'

* * *

ELI LILLY MEDICAL RESEARCH FELLOWSHIP (SOUTH AFRICA)

ESTABLISHED BY THE CAPE TOWN POST-GRADUATE MEDICAL ASSOCIATION

1. Applications are invited from suitably qualified medical practitioners for the Eli Lilly Medical Research Fellowship (South Africa).
2. The Fellowship is for the purpose of medical research and is not intended for post-graduate clinical study. It is available for one year.
3. The value of the Fellowship is 3,000 United States dollars for one year and, in addition, travelling expenses will be allowed, based on a travel budget to be submitted by the Fellow. This will cover the cost of travel and incidental expenses from the place of residence of the Fellow to the approved place of study in the United States of America, as well as the return journey.
4. Other things being equal, preference will be given to candidates under 40 years of age.
5. Any medical practitioner *registered* in South Africa will be eligible for this award.
6. There will be no discrimination for the award on grounds of race, colour, creed or sex.
7. The candidate must submit evidence of his capacity to do original research work.
8. The candidate must submit a programme of the proposed research. He is advised to submit an alternative scheme in case there are difficulties about carrying out the first one.
9. It is advisable for the candidate to indicate at what institution he proposes to undertake the research and he should also state whether he is in a position to make any arrangements to carry out the research at the proposed institution.
10. The successful candidate must undertake to return to South Africa for a period of at least two years after the termination of the award.
11. Applications should be forwarded to reach:

Dr. H. A. Shapiro (Honorary Chairman), Selection Committee, Eli Lilly Medical Research Fellowship (South Africa), P.O. Box 1010, Johannesburg, not later than 30 April 1956.

They should be concise, and accompanied by the names of not more than two suitable referees. Testimonials must not be included.

2 January 1956.

BOOK REVIEW

CONGENITAL HEART DISEASE

Diagnosis of Congenital Heart Disease: A Clinical and Technical Study by the Cardiologic Team of the Pediatric Clinic Karolinska Sjukhuset, Stockholm. By Sven R. Kjellberg, Edgar Mannheimer, Ulf Rudhe and Bengt Jonsson. Pp. 649+xv. With 581 figures $22.) 1955. Chicago: The Year Book Publishers, Inc.

The contents of this strikingly designed volume on the diagnosis of congenital heart disease are based on clinical and radiographic studies of almost 400 cases investigated at the Paediatric Clinic of Karolinska Sjukhuset, Stockholm, from 1951 to 1954.

Excellent chapters dealing with the normal embryology of the heart, its X-ray appearances and physiology, are followed by descriptions of the various investigations undertaken to establish a diagnosis. The extensive use of this great number of special investigations apparently served in assessing the relative value of the various methods.

Routine investigations including conventional radiography, phonocardiography, electrocardiography, electrokmyography, cardiac catheterization and angiocardiography are described in the first 3 chapters.

Angiocardiography, which is always performed under general anaesthesia, aids the surgeon in his assessment of the operability and the appropriate surgical technique, and elucidates such matters as the site and extent of valvular stenosis, the size and localization of a septal defect, the degree of over-riding of the aorta, and the appearance of the systemic arteries and the pulmonary vessels. Simultaneous lateral and frontal views are obtained and, when necessary, exposures can be made at the rate of 12 per second. Simultaneous electrocardiography will help to fix the exact time in the cardiac cycle at which the exposure was made.

A discussion of specific cardiac abnormalities follows the introductory chapters. A general description of the clinical features and physical signs of pulmonary stenosis includes the observation that in Sweden, where the majority of children are under continuous health control from birth onwards, the loud systolic murmur leads to the diagnosis of these cases at a very early age. In almost every instance the condition was discovered in the course of a routine medical examination, as there are no marked symptoms in early life.

It is possible to distinguish valvular from infundibular pulmonary stenosis by simple auscultation, phonocardiography allowing this differentiation to be made with greater certainty. A great deal of investigation has been done on electrokymograms recorded over the pulmonary artery and the right atrium, though the value of this is still very doubtful, as it requires a very accurate positioning of the photo-electric cell.

The angiocardiograms in this section are of a very high standard, and in many cases the autopsy appearances are contrasted with the angiocardiograms. One film is particularly instructive, as it shows a post-operative aneurysm in the right ventricle. Similar dilatations occur while the contrast medium is being injected.

The chapter on the tetralogy of Fallot illustrates that pulmonary stenosis and atresia are merely a question of degree as far as function is concerned. The tetralogy of Fallot is the most common cardiac malformation associated with cyanosis in which the patients survive the first years of life. The abnormality occurred in 38 of the 400 cases, and in 4 cases atresia of the pulmonary artery was present, the blood supply to the lungs being via other channels. The outlook in these cases is particularly poor.

The authors stress the importance of selective angiocardiography in their technique of simultaneous frontal and lateral projections. The contrast-filled right atrium overlaps certain parts of the outflow tract of the right ventricle, hence the importance of injecting this medium directly into the right ventricle. They report that in all but one of their cases the stenosis or atresia of the infundibulum could be visualized, and excellent films support this statement.

The determination of the degree of over-riding of the aorta is important. Contrast medium in the right ventricle may pass through a small septal defect due to the rise in pressure in the right ventricle during the injection of the opaque medium, and this would suggest a greater degree of over-riding than actually exists.

The abnormality of ventricular septal defect is another common form of congenital heart disease. Special attempts have been made to determine whether the defect changes with the growth of the patient. The answer to this remains uncertain, and only follow-up examinations will indicate whether or not the defect becomes obliterated with growth.

Angiocardiograms are also demonstrated of a case in which the defect in the membranous part of the ventricular septum, combined with pulmonary stenosis, was further complicated by the presence of a patent ductus arteriosus.

In cases of atrial septal defect, the authors consider that the systolic murmur does not occur on shunting of the blood through the defect, but is caused by the increased blood flow through the pulmonary artery. The murmur is louder when there is coincident pulmonary stenosis.

A cardiac catheter may pass from the right to the left atrium through a non-functioning but patent foramen ovale. A left-to-right shunt may be demonstrated at the same time, but may be due to the opening of a pulmonary vein into the right atrium. This defect can be distinguished by the use of a special balloon-tipped catheter.

Angiocardiography is a routine investigation in all cases of suspected coarctation of the aorta, but is performed through a Cournand or Lehman catheter into the ascending aorta from the radial artery.

The clinical and the special findings of a variety of other congenital cardiac abnormalities are described. The highly specialized investigations in all these cases are well documented, analysed and illustrated.

This volume is essentially a record of special investigations, and will be of inestimable value to all physicians interested in cardiology. There are 581 X-ray reproductions, diagrams and recordings in the 649 pages. The reproductions are clear and well tabulated, and this book is highly recommended to all who wish to know the scope of special investigations in congenital heart disease.

South African Medico-Legal Society

P.O. BOX 6434 ● JOHANNESBURG

The object of this Society is the promotion of medico-legal knowledge in all its aspects.

This is attained *inter alia* by holding meetings at which papers are read and discussed.

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To The Honorary Secretaries,
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Johannesburg.

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I enclose my cheque for £2. 2. 0, being the subscription for 1 year.

Eli Lilly Medical Research Fellowship (South Africa)

Applications are invited for the 1956 award

of this Fellowship

See p. 91 of this issue for detailed conditions.

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